

INTERACTION OF ALCOHOL AND OTHER DRUGS

An Annotated Bibliography



ADDICTION RESEARCH FOUNDATION
Toronto, Ontario, Canada



Digitized by the Internet Archive
in 2017 with funding from
University of Toronto

<https://archive.org/details/interactionofalc00pola>

INTERACTION of ALCOHOL AND OTHER DRUGS

An annotated Bibliography

Addiction Research Foundation Bibliographic Series

This series is published as a service to scholars concerned with problems of alcohol and drug use. The bibliographies can be obtained by writing to the Documentation Department, Addiction Research Foundation, 33 Russell Street, Toronto 179, Ontario, Canada. Bibliographies 1-4 are now available, and the others are in preparation.

- | | |
|--|------------------------|
| No. 1 <i>Culture and Alcohol Use. A Bibliography of Anthropological Studies.</i> | (Published 1967) |
| No. 2 <i>An Interim Guide to the Cannabis (Marihuana) Literature.</i> | (Published 1968) |
| No. 3 <i>Interaction of Alcohol and Other Drugs. Second Edition, Revised.</i> | (Published 1972) |
| No. 4 <i>Non-Alcoholic Drugs and Personality: An Annotated Bibliography.</i> | (Published 1972) |
| No. 5 <i>Solvent Abuse.</i> | (To be published 1972) |
| No. 6 <i>Teratogenic Effects of Psychoactive Drugs.</i> | (To be published 1972) |
| No. 7 <i>Cannabis: A comprehensive Bibliography.</i> | (To be published 1973) |

Editor of the Series: E. Polacsek
Documentation Department
Addiction Research Foundation
Toronto 179, Ontario
Canada

INTERACTION OF ALCOHOL AND OTHER DRUGS

Second Edition, Revised

An annotated bibliography of the scientific literature on the interaction of ethanol and other chemical compounds normally absent in vivo, the influence of congeners in alcoholic beverages, conjunctive addiction to ethanol plus other drugs, and cross-tolerance between ethanol and other compounds.

Compiled at the

ADDICTION RESEARCH FOUNDATION DOCUMENTATION DEPARTMENT

by

E. POLACSEK, T. BARNES, N. TURNER, R. HALL, AND C. WEISE

With a Foreword by MARK KELLER



Published and Distributed by the

ADDICTION RESEARCH FOUNDATION

Toronto, Ontario, Canada



Z
7721
P58
1972

CONTENTS

Foreword by Mark Keller	vii
Preface	ix
Introduction	xi
Bibliography	1
Key Word Index	501
Author Index	521
Drug Index	539

FOREWORD

The fact that it is becoming nearly impossible to keep up with the flooding flow of information in many fields is by now well known. Some scientists have reached a point of quiescent discouragement. No longer hoping to keep up, they are reconciled to living with the limited random access provided by a few narrowly specialized periodicals. Others are content to delude themselves that they need only to skim "the latest news," and they are served by newly forming "information systems" or "clearinghouses" which cater to this shortsighted perspective. Society and science both need more.

Contemporary society is distressed by an increasing complexity of old problems—often giving the impression of being new when seen in the absence of historical perspective. Thus, people commonly imagine that environmental pollution is a "modern" or recently developed problem caused by technology, or overpopulation or affluence. Actually, the early agriculturists and herders already caused environmental deterioration and experienced its effects as they too achieved advances in technology, growth of population and gains in affluence. It is likely that the contemporary difficulty could be better understood, that there might be less discord about causes and possible remedies, if the problem-complex were viewed wholistically and in light of the inherent biosocial characteristics of man and his multifarious motivations.

Mood-altering drugs are not modern. It is curious that the problems they engender should be attributed to industrialization or advertising or publicity, despite ample evidence of their existence throughout recorded history and even earlier. Nothing should be more obvious than that the use of such drugs by man is related to elemental aspects of his nature, of his intricate physical, psychic and social inventory.

That a problem is old does not reduce the need to try to cope with it. Even if the cause does not lie in the greater complexity of contemporary life, that circumstance intensifies its pains and costs. One effect of this is a growth of study and reporting by the disciplines and professions to which the society now looks for knowledge and alleviation. But the parallel of increasingly complex social organization is an increased specialization of functions. Not only do entirely different people bake bread, typewrite letters, design clothes, drive taxis, heal wounds, teach dancing or conduct funerals, but different people study chemistry or biochemistry, psychology, virology, economics, or political science. When society turns to the scientific-academic community for the expertise needed to gain relief from some distressing problem—whether environmental pollution by technological by-products or personality dissolution by addictive products—it gets not all-encompassing knowledge, but a melange of particularistic answers. Psychiatrists and educators, psychologists and theologians, penologists and anthropologists, biochemists and physicians, all have some specialized knowledge, and often each discipline and profession imagines that its information is sufficient for society's needs—whether to remedy or to prevent. This is a penalty of the benefits of specialization. However, none of the specialist groups holds an adequate answer to such a complicated problem as drug misuse; in fact, not even for any one aspect of "the problem." Thus, the biochemists alone do not know enough to explain the biochemical phenomena—for, so complex is man, that psychological events and social circumstances influence body chemistry. The knowledge needed to understand the multi-problem, then, must come from a variety of sources. But it can hardly be used effectively unless it is brought together in such a way that capable minds can evaluate and analyze and synthesize it.

The process of bringing together is the specialism of a new profession of documentation—some choose to call it information science. An inevitable feature of a lately arisen informationism is the immediate immersion in computerism with the hasty output of current informational bits in response to desperate demands for "the answer." The currentness is often touted as an advantage, though the real reason for concentrating on it may be nothing else

than the lack of resources to tap the treasures of the past: it is cheapest and easiest to start any information system with the reportage of the start-up year. Time and experience will remedy the errors of prematurity and overpromotion. But fortunately these errors are not universal. Already there are information specialists in some fields who have insightfully sought to produce a more sophisticated management of the existing knowledge. The present work is an example of the latter sort. A thoughtfully designed and richly indexed compilation of bibliography and abstracts, it focuses on the problems of the use of drugs by people, and especially combinations of drugs, in ways that threaten personal and societal harms. The materials have been gleaned from every possible source of knowledge, not from any limited disciplinary or professional or linguistic or geographic area. And full advantage has been taken of the opportunity for historical coverage. This not only gives access to the experience of the past, so that duplication of research and theorizing can be averted, but allows also the broad perspective which is essential if, some time, we are to achieve the sort of synthesis which allows deep understanding and can lead to fundamental alleviations.

This, then, is a documentation of superior caliber, offering the worlds of scholarship and action an invaluable tool basic to education and progress. Although addressed to the interactions of drugs, it really goes further: it covers the interactions of people with drugs.

One thing is regrettable: that the coverage is limited to a selection of drugs rather than all of them. This is not said carpingly. The limitation is necessitated by problems of time and resources, not by lack of interest or awareness. But it is better to have restricted the range of drugs and cover them as deeply as this work does, than to have chosen all-inclusiveness but with inferior quality of treatment. We are fortunate to have this first-rate documentation of the interactions of that most popular drug, alcohol, with the other ubiquitous drugs of harmful misuse. The rest can be done in due course and will be done, no doubt, with the same intensity and devotion to excellence.

Mark Keller

PREFACE

The second edition of *Interaction of Alcohol and Other Drugs* marks the close of a period of bibliographical research and development, and the beginning of a phase of publication of “new style”, regularly updated, computer-produced bibliographies in the field of research on non-medical drug use. What James Atkinson, in his *Medical Bibliography* of 1834, called “the dry, dusty, tedious, accursed, hateful bibliography” has, with the aid of computer technology, hopefully been transformed into a more dynamic and current reference tool. The supplements to the present edition, and all subsequent works produced by this method, can thereby be published within a relatively short time after input of the last desired item of information into the computer data base, without the necessity for typesetting or additional proof-reading.

This edition completes, as far as possible, all retrospective searching on the subject up to the last several years. For the most recent period, coverage is extensive but not exhaustive; however, new material will be found in the forthcoming supplements, and it is expected that the degree of currentness will increase in these later publications as the operation itself is further refined and streamlined.

In compiling a work of this nature, it has been fortunate that advice was available to us from a number of experts within the Addiction Research Foundation, although a variety of helpful comments and suggestions were received as well from readers of the First Edition. We are especially grateful to Dr. Harold Kalant, Dr. Eugene LeBlanc, and Dr. Rosemary Hawkins for their valuable critical discussion of the definition and scope of the material during its preparation. In addition, the interest and guidance of Mr. Jan DeLint and Dr. Wolfgang Schmidt have played a significant role throughout the progress of this work, and have been warmly appreciated. A special tribute is also owed to Mr. Robert Popham, Head of the Research Division, who founded the Addiction Research Foundation Bibliographic Series, and whose continued support has enabled the present volume and the computerized operation upon which it is based to be brought to completion.

INTRODUCTION

Aim of the Bibliography

The aim of this bibliography is to give as comprehensive as possible a coverage of all scientific papers published in any language on the subject of alcohol interactions. Full bibliographical citations, abstracts, and indexing are shown for each item. Considerable care has been taken to provide complete references, firstly to enable readers to obtain items of interest easily, and, secondly, to enable the bibliography to be used as a standard reference source.

The present edition includes information on papers published in twenty languages. Approximately 45% of these papers have been written in languages other than English, some of which may have been previously unknown or inaccessible to many readers. In this area of research, as in most others, it is quite apparent that writers tend to a large degree to restrict literature searching to works published in their respective native languages, and, hence, it is to be hoped that the bibliography may in some way serve to internationalize further the research in this field.

The present edition is also intended to form the foundation for a continuously supplemented, regularly updated coverage of the literature. To this end, a computer program has been designed which allows the regular input of current bibliographical data, and easier publication of supplements at desired intervals. The present bibliography is the first publication to be computer-printed through use of the program. By means of this updating, it is intended that the bibliography on alcohol interactions will remain current and of continuing value to users.

Scope and Extent of Coverage

The range of literature contained includes research studies, clinical reports, review articles, papers concerned with medico-legal implications of ethanol-drug interactions, and commentaries of all kinds, including letters to the editor. In compiling the bibliography, the position has been adopted that evaluation in a complex work of this nature should and must rest with the reader. Therefore, generally speaking, no evaluation of any article has been made, and all scientific papers, in any language, which discuss the subject directly or indirectly have been included.

Retrospective literature coverage has been made back to the earliest-known paper. It cannot be denied that some, at least, of the earlier studies undertaken in this area have substantial relevance to present-day research. Mark Keller, in his introduction to Volume 1 of the *International Bibliography of Studies on Alcohol*, aptly comments on this point:

If. . . [the researcher's] information is confined by language or geography, his contemplated experiment may already have been done in another country. If his sources are narrowed by time or by class of periodical, his contemplated experiment may have been done twenty years ago, or reported in the literature of another profession. We have all seen this waste, that able people repeat work already accomplished, rediscover answers already known, because the reports from another country or time or sphere of work were not available—alas, sometimes not sought—when they could have devoted their thought and energy and resources to the next step. . .

. . . Even as this introduction is written, an article is in press reporting on the newest and most modern form of treating alcoholics by the use of electric current. It seems a pity that the author and deviser of this treatment has no knowledge of an article published over half a century ago discussing the same idea. The barrels of blood and rivers of urine that will yet be collected

from alcoholic and other patients to determine already reported contents are immeasurable and not worth the effort. Yes, there is information—not only in the biological realm—and wisdom, too, in the older writings; and it would be far more economical to make them accessible by resourceful documentation than to recapitulate the learning.

Subjects Included in the Bibliography

A. Interaction of Ethanol With Other Drugs

Included were papers dealing with the following:

1. Interaction of ethanol with other compounds (usually, but not exclusively, those which are not normal body constituents), and the effects of this interaction upon the functioning (physiological, biochemical, and behavioural) of intact humans, animals, and micro-organisms, and upon isolated functional systems, both cellular and sub-cellular.
2. Clinical reports which concentrate mainly on short-term treatment of the acute alcoholic situation—e.g., acute intoxication, acute poisoning, and alcoholic coma.
3. Unexpected side-effects occurring in conjunction with alcohol consumption during drug therapy.
4. Studies concerning tests to determine blood alcohol levels, if substances purportedly influencing the blood alcohol level were involved.
5. Drug-induced sensitization or intolerance to alcohol, with respect to reactions resulting from accidental exposure to a sensitizing compound, or to unexpected side-effects of drug treatment for a given medical condition.

Excluded were papers concerning:

1. The influence of other compounds on secondary physiological consequences of ethanol ingestion (e.g., effects of alkalis on ethanol-induced acidosis).
2. Long-term treatment of alcoholism.
3. The influence, on blood alcohol measurements, of substances which cause false positive determinations only (in contrast to actual reported influencing of the blood alcohol level).
4. Sensitization drugs commonly used for alcohol therapy (e.g., disulfiram), except when other drugs are also mentioned.

B. Congeners in Alcoholic Beverages

Mainly, but not exclusively, included were studies which compared congener beverages to pure ethanol solutions or vodka.

C. Conjunctive Addiction to Ethanol Plus Other Drugs

D. Cross-Tolerance Between Ethanol and Other Drugs

Types of Information Provided in the Bibliography

A. References

Titles are listed alphabetically according to the senior author. The complete information for every title is given, including the number of references cited by the authors. The titles of non-English language articles are given in the original language, followed by an English translation in square brackets. The names of authors and the titles of papers published in the two non-Latin alphabet languages included in the bibliography, Russian and Japanese, have been transliterated—the former according to the Library of Congress System (diacritical marks omitted), and the latter according to the Hepburn System. Titles of monographs, collected or edited works, dissertations, and unpublished papers are in italics; journal articles and book chapters are in regular type. Titles of all journals included in the *Index Medicus Abbreviation Listing* (1969) are abbreviated according to this system. All other journal titles are given in full; for the sake of consistency, the capitalization of the titles in this latter group, including non-English language journals, has been conducted according to standard English-language practice. The name of the place of publication is listed, foreign place names being given in the English-language version. North

American theses are referred to as M.A. or Ph.D. theses, whereas foreign graduate works are referred to as dissertations. The country of origin of German dissertations dated earlier than 1945 is listed as Germany, and after that date as East or West Germany. If duplicate articles have been published in several journals, they have been treated as separate articles, the same abstract being used for identical texts.

B. *Key Word Terms*

Following each citation are abbreviated key word index terms which provide supplementary information, which may or may not be mentioned in the abstract, such as: language of publication, type of paper (e.g., experimental, review, etc.), subject area of the cited paper (e.g., conjunctive addiction, medico-legal, etc.), kinds of experimental subjects used, anatomical components or physiological processes affected by interaction, broad subject groupings of compounds listed which interact with ethanol, and *Classified Abstract Archive of the Alcohol Literature* abstract number and position, if applicable. See the *Key Word Abbreviation List* for a full outline of the 82 terms of the Special Index.

C. *Accession Numbers*

Because it is hoped, resources and demand permitting, to put the full text of items on file at the Addiction Research Foundation onto microfiche, each paper contained in the bibliography has been assigned a permanent, unique accession number (e.g., A-1327, B-0250), whereby it can easily be identified.

D. *Abstracts*

Abstracts of approximately 150-200 words are given for each item cited. Every effort was made to provide as much information as possible, but it should be emphasized that the abstracts are primarily intended to allow the user to make a negative selection, and to reject with reasonable certainty those papers which are not relevant for his purpose. Nevertheless, in response to suggestions of readers of the First Edition, an effort has been made to provide more extensive and informative abstracts for the 450 additional items included in the Second Edition. Since the abstracts concentrate almost exclusively on the alcohol interaction aspect of each paper, a "secondary article" (SEC) key word term has been applied to some bibliographical items, in order that the reader will not be misled in the case of abstracts of papers in which the main aim has not been to investigate or discuss the interaction of ethanol with other compounds, but in which some oblique reference to combined effects is made, or else the space relevant to ethanol interaction is a relatively small part of the whole text. The "SEC" term, therefore, signifies only that the major concentration of a paper is on a non-interaction aspect; it does not necessarily indicate that the item is of lesser significance to the subject of alcohol interactions.

E. *Key Word Index*

The Key Word Index is a cumulated listing of all items to which each of the 82 key word terms has been applied.

F. *Author Index*

All senior and junior authors are listed, together with the numbers of the titles of papers published by them. Where the author is a senior author, the title numbers appear in bold type. For technical reasons, it has been found necessary to list only the initials of the first and middle names of each author, regardless of the form in which the name appears in the original paper. Although it is true that the text of the bibliography is organized alphabetically according to senior author names, this separate index has nevertheless been found necessary—in order to permit, for example, easy location of authors (who may well be, of course, more "senior" in fact) other than the senior authors.

G. *Index of Interaction Drugs*

The drug index is a list of all individual drug names mentioned in the papers, with reference to alcohol-drug interaction. If more than 12 drug names are contained in a given paper, only the corresponding drug groups are listed. However, if an article reports on a specific experiment, all the drugs used in the experiment are listed, whatever the number.

The drug names are cross-referenced according to information obtained from the following standard works: E.E.J. Marler, *Pharmacological and Chemical Synonyms*, 4th ed. (Amsterdam: Excerpta Medica Foundation, 1967); M. Negwer, *Organisch-chemische Arzneimittel und ihre Synonyma* (Berlin: Akademie-Verlag, 1966); and P. G. Stecher, ed., *The Merck Index*, 8th ed. (Rahway, N. J.: Merck and Co., Inc., 1968). In the code used for the index, one of the letters listed below appears in bold type after the drug name:

- R — recommended International Non-proprietary Name (W.H.O.)
P — proposed International Non-proprietary Name (W.H.O.), British Approved Name, or United States Approved Name
C — chemical and/or common name
N — drug name which could not be verified in any of the above reference works

In cross-referencing, the recommended International Non-proprietary Name (R) is preferred as the standardized form, while the proposed International Non-proprietary Name (P) is the second choice, the chemical and/or common name (C) is the third choice, and the unverified name, naturally, is fourth. The numbers of entries in the bibliography text are listed only under the most-preferred, or highest-choice drug name.

If possible, combination drugs have been verified in C. O. Wilson and J. E. Jones, eds., *American Drug Index* (Philadelphia: J. B. Lippincott, 1964). If a combination drug is not listed in this source, the composition has been determined from the article itself, and is given in parentheses. A plus sign (+) following the last component indicates that not all parts of the composition could be found.

Abbreviations Used

A. Languages

A — Afrikaans	J — Japanese
C — Czech	N — Norwegian
D — Dutch	P — Portuguese
Da — Danish	Po — Polish
E — English	R — Russian
F — French	Ru — Rumanian
Fi — Finnish	S — Swedish
G — German	Se — Serbo-Croatian
H — Hungarian	Sp — Spanish
I — Italian	T — Turkish

B. Summaries in Other Languages

All foreign language papers with a summary in a second language are indicated, as in the examples below:

- ES — English summary
FS — French summary
GS — German summary

C. Key Word Term Abbreviations

Qualification of Paper

1. SEC

secondary article (article in which the main aim is not to investigate or discuss the interaction of ethanol with other compounds, but some oblique reference to combined effects is made, or else the space pertinent to ethanol interaction is a relatively small part of the whole text)

Type of Paper

2. abst.

abstract only

3. exp. cont.

experimental study, controlled

4. exp. comp.

experimental study, comparative

5. exp.

experimental study, other than items 3 and 4

6. general

general paper, with no original experimental or statistical research

7. presentation

presentation, published or not published in a regular journal, made before a conference, seminar, or session of an organization

8. review

review of literature

9. stat. surv.

survey or statistical research

Subject of Paper

10. case hist.

case histories (concerning treatment of intoxication or toxic effects)

11. congen. stud.	<i>congener studies</i>
12. conj. addict.	<i>conjunctive addiction (addiction to alcohol plus other drugs)</i>
13. cross-tol.	<i>cross-tolerance</i>
14. DC (antidotal)	<i>drug combinations—antidotal use in clinical treatment (exclude item 15)</i>
15. DC (decrease)	<i>drug combinations—decrease of drug effects or of toxic effect on organism (exclude item 14)</i>
16. CD (supra-add. incr.)	<i>drug combinations—supra-additive (more than additive) increase of drug effects or of toxic effect on organism (if specifically stated as such in the paper)</i>
17. DC (add., infra-add., unspec. incr.)	<i>drug combinations—additive, infra-additive (less than additive), or unspecified increase of drug effects or of toxic effect on organism</i>
18. DC (unchanged)	<i>drug combinations—unchanged drug effects</i>
19. DC (unspec.)	<i>drug combinations—unspecified drug effects</i>
20. DC (sensit.)	<i>drug combinations—sensitization or intolerance to alcohol</i>
21. med.-leg.	<i>medico-legal</i>
22. mot. vehic.	<i>operation of motor vehicles or simulators—including all forms of surface and air motor transport</i>
23. post.-mort.	<i>post-mortem findings in interaction poisonings</i>

Subjects or Beings Affected by Interaction

24. humans	<i>humans, healthy or to whom items 25 and 26 do not apply</i>
25. psychot. humans	<i>humans, psychotic or labile</i>
26. drug-dep. humans	<i>humans, drug-dependent or drug-dependence-prone</i>
27. mammals	<i>mammals (excluding humans)</i>
28. other org.	<i>other organisms</i>

Drug Administration in Experimental Studies

29. acute admin.	<i>acute administration</i>
30. chronic admin.	<i>chronic administration</i>
31. in vivo	<i>in vivo</i>
32. in vitro	<i>in vitro</i>

Results of Drug Intake in Experimental and Non-Experimental Papers

33. dose resp.	<i>dose response curve</i>
34. blood lev.	<i>levels of drugs in blood</i>
35. other drug lev.	<i>levels of drugs, other than in blood (urine, breath, sweat, saliva, faecal levels)</i>
36. mot. perform.	<i>motor performance</i>
37. psychol. perform.	<i>psychological performance</i>
38. species or sex. diff.	<i>species or sex differences in response</i>

Anatomical Components or Physiological Processes Affected by Interaction

39. absorp., distrib., stor.	<i>absorption, distribution, or storage</i>
40. acid-base, blood pH, elect.	<i>acid-base balance, blood pH, electrolytes</i>
41. blood comp., sites, lymph	<i>blood components (blood cells, platelets, plasma), cell-forming sites, and lymph</i>
42. cardiovasc.	<i>cardiovascular system (heart, blood vessels, blood circulation)</i>
43. CNS	<i>central nervous system</i>
44. G.I. tract	<i>gastro-intestinal tract, its glands and secretions</i>
45. glands	<i>glands (endocrine and exocrine) and their secretions, except items 44 and 46</i>
46. liver, kidney	<i>liver, kidney (including urine)</i>
47. metab. proc.	<i>metabolic processes</i>

48. nerv. syst.	<i>nervous system, other than CNS</i>
49. respir.	<i>respiration, including breath</i>
50. senses	<i>senses and sensation</i>
51. skel., muscle, skin	<i>skeleton, muscle, skin</i>

Compounds Interacting With Ethanol

52. alcohols	<i>alcohols, other than ethanol, such as methanol, propanol, etc.</i>
53. amphetamines	<i>amphetamines (dexamphetamine, methamphetamine, etc.)</i>
54. analeptics	<i>analeptics (bemegride, meclogenoxate, etc.)</i>
55. analg., antipyret.	<i>analgesics and antipyretics (APC, phenacetin, etc.)</i>
56. anesthetics	<i>anesthetics (butamin, ether, cocaine, etc.)</i>
57. anticonvulsants	<i>anticonvulsants (carbamazepine, phenytoin, etc.)</i>
58. antidepressants	<i>antidepressants (desimipramine, prolintane, etc.)</i>
59. anti-infectants	<i>anti-infectants (penicillin, cycloserine, etc.)</i>
60. antispasmodics	<i>antispasmodics (ethyl acetate, papaverine, etc.)</i>
61. autocoids	<i>autocoids (antihistamine, clemizole, etc.)</i>
62. autonomic agents	<i>autonomic agents (parasympatholytics, parasympathomimetics, sympatholytics, sympathomimetics)</i>
63. barbiturates	<i>barbiturates (barbital, secobarbital, etc.)</i>
64. coagulants	<i>blood formation and coagulation agents, or anticoagulation agents (oxalic acid, phenprocoumon, etc.)</i>
65. cardiovasc. agents	<i>cardiovascular agents (digitalis, dopamine, etc.)</i>
66. diagnost. agents	<i>diagnostic agents (sodium benzoate, sodium iodide, etc.)</i>
67. elect., water-bal. agents	<i>electrolyte and water-balance agents (diuretics, ions, glucose, etc.)</i>
68. enzymes	<i>enzymes (monoamine oxidase inhibitors, iproniazid, etc.)</i>
69. gastrointest. agents	<i>gastrointestinal agents (meclozine, glutamic acid, etc.)</i>
70. hallucinogens	<i>hallucinogens (cannabis, lysergide, etc.)</i>
71. hormones, hormone antag.	<i>hormones and hormone antagonists (ACTH, insulin, etc.)</i>
72. indust. intox.	<i>industrial intoxicants (carbon monoxide, gasoline, etc.)</i>
73. integ. syst. agents	<i>integumentary system agents (camphor, menthol, etc.)</i>
74. miscellaneous	<i>miscellaneous compounds used in manufacturing or not otherwise specified</i>
75. musculoskel. agents	<i>musculoskeletal agents (antirheumatics, muscle relaxants, etc.)</i>
76. neoplast. agents	<i>neoplastic agents (alloxan, urethane, etc.)</i>
77. nutritive agents	<i>nutritive agents (vitamins, etc.)</i>
78. respir. agents	<i>respiratory agents (potassium iodide, tyloxapol, etc.)</i>
79. sed., hypnot.	<i>sedatives and hypnotics (excluding barbiturates) such as apronal, glutethimide, etc.</i>
80. stimulants	<i>stimulants, excluding amphetamines (pemoline, caffeine, etc.)</i>

D. Journal Title Abbreviations

The following abbreviations, based on *Index Medicus*, 1969, appear in the bibliography:

Acta Med.	<i>Acta Medica (Igaku Kenkyu) (Fukuoka)</i>
Acta Med. Jugosl.	<i>Acta Medica Jugoslavica (Belgrade)</i>
Acta Med. Scand.	<i>Acta Medica Scandinavica (Stockholm)</i>
Acta Morph. Acad. Sci. Hung.	<i>Acta Morphologica Academiae Scientiarum Hungaricae (Budapest)</i>
Acta Neurol. Belg.	<i>Acta Neurologica et Psychiatrica Belgica (Brussels)</i>
Acta Otolaryng.	<i>Acta Oto-laryngologica (Stockholm)</i>
Acta Pharmacol.	<i>Acta Pharmacologica et Toxicologica (Copenhagen)</i>
Acta Physiol. Lat. Amer.	<i>Acta Physiologica Latino-Americana (Buenos Aires)</i>
Acta Physiol. Pol.	<i>Acta Physiologica Polonica (Warsaw)</i>

Acta Physiol. Scand.	<i>Acta Physiologica Scandinavica (Stockholm)</i>
Acta Soc. Med. Upsal.	<i>Acta Societatis Medicorum Upsaliensis (Stockholm)</i>
Actas Luso Exp. Neurol. Psiquiat.	<i>Actas Luso-Espanolas de Neurologia y Psiquiatria (Madrid)</i>
Activ. Nerv. Sup.	<i>Activitas Nervosa Superior (Prague)</i>
Aerospace Med.	<i>Aerospace Medicine (St. Paul)</i>
Amer. Ass. Industr. Nurses J.	<i>American Association of Industrial Nurses Journal (Pitman, N. J.)</i>
Amer. Heart J.	<i>American Heart Journal (St. Louis)</i>
Amer. Industr. Hyg. Ass. J.	<i>American Industrial Hygiene Association Journal (Detroit)</i>
Amer. J. Clin. Nutr.	<i>American Journal of Clinical Nutrition (Bethesda)</i>
Amer. J. Clin. Path.	<i>American Journal of Clinical Pathology (Baltimore)</i>
Amer. J. Dig. Dis.	<i>American Journal of Digestive Diseases (New York)</i>
Amer. J. Med.	<i>American Journal of Medicine (New York)</i>
Amer. J. Med. Sci.	<i>American Journal of the Medical Sciences (Philadelphia)</i>
Amer. J. Obstet. Gynec.	<i>American Journal of Obstetrics and Gynecology (St. Louis)</i>
Amer. J. Ophthal.	<i>American Journal of Ophthalmology (Chicago)</i>
Amer. J. Path.	<i>American Journal of Pathology (New York)</i>
Amer. J. Physiol.	<i>American Journal of Physiology (Bethesda)</i>
Amer. J. Proctol.	<i>American Journal of Proctology (New York)</i>
Amer. Rev. Resp. Dis.	<i>American Review of Respiratory Disease (Baltimore)</i>
Anat. Rec.	<i>Anatomical Record (Philadelphia)</i>
Anesth. Analg. (Cleveland)	<i>Anesthesia and Analgesia; Current Researches (Cleveland)</i>
Anesth. Analg. (Paris)	<i>Anesthesie, Aualgesie, Reanimation (Paris)</i>
Angew. Chem.	<i>Angewandte Chemie (Weinheim)</i>
Ann. Chir. Gynaec. Fenn.	<i>Annales Chirurgiae et Gynaecologiae Fenniae (Helsinki)</i>
Ann. Med. Exp. Biol. Fenn.	<i>Annales Medicinae Experimentalis et Biologiae Fenniae (Helsinki)</i>
Ann. Med. Intern. Fenn.	<i>Annales Medicinae Internae Fenniae (Helsinki)</i>
Ann. Med. Leg.	<i>Annales de Medecine Legale (Paris)</i>
Ann. Medicopsychol.	<i>Annales Medico-psychologiques (Paris)</i>
Ann. N.Y. Acad. Sci.	<i>Annals of the New York Academy of Sciences (New York)</i>
Ann. Ottal.	<i>Annali di Ottalmologia e Clinica Oculistica (Parma)</i>
Ann. Pharm. Franc.	<i>Annales Pharmaceutiques Francaises (Paris)</i>
Ann. Surg.	<i>Annals of Surgery (Philadelphia)</i>
Arch. De Vecchi Anat. Pat.	<i>Archivio De Vecchi per l'Anatomia Patologica e la Medicina Clinica (Florence)</i>
Arch. Environ. Health	<i>Archives of Euvironmental Health (Chicago)</i>
Arch. Fisiol.	<i>Archivio di Fisiologia (Florence)</i>
Arch. Gen. Psychiat.	<i>Archives of General Psychiatry (Chicago)</i>
Arch. Int. Pharmacodyn.	<i>Archives Internationales de Pharmacodyuamie et de Therapie (Gand)</i>
Arch. Intern. Med.	<i>Archives of Interual Medicine (Chicago)</i>
Arch. Mal. Prof.	<i>Archives des Maladies Professionnelles, de Medecine du Travail et de Securite Sociale (Paris)</i>
Arch. Maragliano Pat. Clin.	<i>Archivio E. Maragliano di Patologia e Clinica (Genoa)</i>
Arch. Neurol.	<i>Archives of Neurology (Chicago)</i>
Arch. Path.	<i>Archives of Pathology (Chicago)</i>
Arch. Pharm.	<i>Archiv der Pharmazie und Berichte der Deutschen Pharmazeuti-schen Gesellschaft (Weinheim)</i>
Arch. Psychiat. Nervenkr.	<i>Archiv fur Psychiatrie und Nervenkrankheiten vereinigt mit Zeitschrift fur die gesaunte Neurologie nnd Psychiatrie (Berlin)</i>
Arch. Toxik.	<i>Archiv fur Toxikologie; Fuehner-Wielands Sammlung von Vergiftungsfaellen (Berlin)</i>

Arizona Med.	<i>Arizona Medicine (Scottsdale)</i>
Aust. Ann. Med.	<i>Australasian Annals of Medicine (Sydney)</i>
Beitr. Gerichtl. Med.	<i>Beitraege zur Gerichtlichen Medizin (Vienna)</i>
Biochem. Biophys. Res. Commun.	<i>Biochemical and Biophysical Research Communications (New York)</i>
Biochem. J.	<i>Biochemical Journal (London)</i>
Biochem. Pharmacol.	<i>Biochemical Pharmacology (New York)</i>
Biochim. Biophys. Acta	<i>Biochimica et Biophysica Acta (Amsterdam)</i>
Boll. Oculist.	<i>Bollettino d'Oculistica (Bologna)</i>
Boll. Soc. Ital. Biol. Sper.	<i>Bollettino della Societa Italiana di Biologia Sperimentale (Naples)</i>
Brit. J. Addict.	<i>British Journal of Addiction (London)</i>
Brit. J. Anaesth.	<i>British Journal of Anaesthesia (Altrincham)</i>
Brit. J. Industr. Med.	<i>British Journal of Industrial Medicine (London)</i>
Brit. J. Pharmacol.	<i>British Journal of Pharmacology (London)</i>
Brit. J. Psychiat.	<i>British Journal of Psychiatry (London)</i>
Brit. Med. J.	<i>British Medical Journal (London)</i>
Bull. Schweiz. Akad. Med. Wiss.	<i>Bulletin der Schweizerischen Akademie der Medizinischen Wissenschaften (Basel)</i>
Bull. Soc. Franc. Derm. Syph.	<i>Bulletin de la Societe Francaise de Dermatologie et de Syphiligraphie (Paris)</i>
C.R. Acad. Sci. [D]	<i>Comptes Rendus Hebdomadaires des Seances de l'Academie des Sciences; D: Sciences Naturelles (Paris)</i>
C.R. Soc. Biol.	<i>Comptes Rendus des Seances de la Societe de Biologie et de Ses Filiales (Paris)</i>
Cah. Anesth.	<i>Cahiers d'Anesthesiologie (Paris)</i>
Calif. Med.	<i>California Medicine (San Francisco)</i>
Canad. Anaesth. Soc. J.	<i>Canadian Anaesthetists' Society Journal (Toronto)</i>
Canad. J. Biochem.	<i>Canadian Journal of Biochemistry (Ottawa)</i>
Canad. J. Public Health	<i>Canadian Journal of Public Health (Toronto)</i>
Canad. Med. Ass. J.	<i>Canadian Medical Association Journal (Toronto)</i>
Cas. Lek. Cesk.	<i>Casopis Lekarů Ceskych (Prague)</i>
Cesk. Farm.	<i>Ceskoslovenska Farmacie (Prague)</i>
Cesk. Psychiat.	<i>Ceskoslovenska Psychiatrie (Prague)</i>
Clin. Lab.	<i>Clinica y Laboratorio (Saragossa)</i>
Clin. Pharmacol. Ther.	<i>Clinical Pharmacology and Therapeutics (St. Louis)</i>
Clin. Sci.	<i>Clinical Science (London)</i>
Curr. Ther. Res.	<i>Current Therapeutic Research (New York)</i>
Derm. Wschr.	<i>Dermatologische Wochenschrift (Leipzig)</i>
Deutsch. Gesundh.	<i>Deutsche Gesundheitswesen (Berlin)</i>
Deutsch. Med. Wschr.	<i>Deutsche Medizinische Wochenschrift (Stuttgart)</i>
Deutsch. Z. Ges. Gerichtl. Med.	<i>Deutsche Zeitschrift für die Gesamte Gerichtliche Medizin (Berlin)</i>
Dis. Nerv. Syst.	<i>Diseases of the Nervous System (Galveston)</i>
Electroenceph. Clin. Neurophysiol.	<i>Electroencephalography and Clinical Neurophysiology (Amsterdam)</i>
Enzym. Biol. Clin. (Basel)	<i>Enzymologia Biologica et Clinica (Basel)</i>
Europ. J. Pharmacol.	<i>European Journal of Pharmacology (Amsterdam)</i>
Farmakol. Toksik.	<i>Farmakologiya i Toksikologiya (Moscow)</i>
Fed. Proc.	<i>Federation Proceedings (Bethesda)</i>
Fiziol. Zh. S.S.S.R. Sechenov.	<i>Fiziologicheskii Zhurnal S.S.S.R. imeni I.M. Sechenova (Moscow)</i>
Folia Pharmacol. Jap.	<i>Folia Pharmacologica Japonica (Kyoto)</i>
Food Cosmet. Toxic.	<i>Food and Cosmetics Toxicology (Oxford)</i>

Fortschr. Neurol. Psychiat.	<i>Fortschritte der Neurologie, Psychiatrie und Ihrer Grenzgebiete (Stuttgart)</i>
G. Geront.	<i>Giornale di Gerontologia (Florence)</i>
German Med. Monthly	<i>German Medical Monthly (Stuttgart)</i>
G.P.	<i>G.P. (Kansas City)</i>
Helv. Physiol. Pharmacol. Acta	<i>Helvetica Physiologica et Pharmacologica Acta (Basel)</i>
Hoppe Seyler Z. Physiol. Chem.	<i>Hoppe-Seyler's Zeitschrift für Physiologische Chemie (Berlin)</i>
Hyg. Ment.	<i>Hygiene Mentale (Paris)</i>
Indian J. Path. Bact.	<i>Indian Journal of Pathology and Bacteriology (Bombay)</i>
Industr. Med. Surg.	<i>Industrial Medicine and Surgery (Chicago)</i>
Int. Arch. Gewerbepath.	<i>Internationales Archiv für Gewerbepathologie und Gewerbehygiene (Berlin)</i>
Int. J. Cancer	<i>International Journal of Cancer (Copenhagen)</i>
Int. J. Neuropsychiat.	<i>International Journal of Neuropsychiatry (Chicago)</i>
Int. Rev. Neurobiol.	<i>International Review of Neurobiology (New York)</i>
Invest. Urol.	<i>Investigative Urology (Baltimore)</i>
J.A.M.A.	<i>Journal of the American Medical Association (Chicago)</i>
J. Amer. Geriat. Soc.	<i>Journal of the American Geriatrics Society (Baltimore)</i>
J. Amer. Med. Wom. Ass.	<i>Journal of the American Medical Women's Association (Nashville)</i>
J. Amer. Pharm. Ass.	<i>Journal of the American Pharmaceutical Association (Washington)</i>
J. Biol. Chem.	<i>Journal of Biological Chemistry (Baltimore)</i>
J. Clin. Invest.	<i>Journal of Clinical Investigation (Boston)</i>
J. Clin. Pharmacol.	<i>Journal of Clinical Pharmacology and the Journal of New Drugs (New York)</i>
J. Comp. Physiol. Psychol.	<i>Journal of Comparative and Physiological Psychology (Washington)</i>
J. Dent. Res.	<i>Journal of Dental Research (Chicago)</i>
J. Forensic Sci. Soc.	<i>Journal of the Forensic Science Society (London)</i>
J. Genet. Psychol.	<i>Journal of Genetic Psychology (Provincetown)</i>
J. Indiana Med. Ass.	<i>Journal of the Indiana State Medical Association (Indianapolis)</i>
J. Kansas Med. Soc.	<i>Journal of the Kansas Medical Society (Topeka)</i>
J. Lab. Clin. Med.	<i>Journal of Laboratory and Clinical Medicine (St. Louis)</i>
J. Louisiana Med. Soc.	<i>Journal of the Louisiana State Medical Society (New Orleans)</i>
J. Med. Bordeaux	<i>Journal de Medecine de Bordeaux et du Sud-Ouest (Bordeaux)</i>
J. Nat. Med. Ass.	<i>Journal of the National Medical Association (New York)</i>
J. Nerv. Ment. Dis.	<i>Journal of Nervous and Mental Disease (Baltimore)</i>
J. Nutr.	<i>Journal of Nutrition (Philadelphia)</i>
J. Obstet. Gynaec. Brit. Comm.	<i>Journal of Obstetrics and Gynaecology of the British Commonwealth (London)</i>
J. Occup. Med.	<i>Journal of Occupational Medicine (New York)</i>
J. Personality Soc. Psychol.	<i>Journal of Personality and Social Psychology (Washington)</i>
J. Pharm. Pharmacol.	<i>Journal of Pharmacy and Pharmacology (London)</i>
J. Pharm. Sci.	<i>Journal of Pharmaceutical Sciences (Washington)</i>
J. Pharmacol. Exp. Ther.	<i>Journal of Pharmacology and Experimental Therapeutics (Baltimore)</i>
J. Physiol. (London)	<i>Journal of Physiology (London)</i>
J. Physiol. (Paris)	<i>Journal de Physiologie (Paris)</i>
Jap. J. Pharmacol.	<i>Japanese Journal of Pharmacology (Kyoto)</i>
Klin. Wschr.	<i>Klinische Wochenschrift (Berlin)</i>
Lab. Invest.	<i>Laboratory Investigation (New York)</i>
Life Sci.	<i>Life Sciences (Oxford)</i>

Maryland Med. J.	<i>Maryland State Medical Journal (Baltimore)</i>
Med. Ann. D.C.	<i>Medical Annals of the District of Columbia (Washington)</i>
Med. Clin. N. Amer.	<i>Medical Clinics of North America (Philadelphia)</i>
Med. Exp.	<i>Medicina Experimentalis (Basel)</i>
Med. J. Aust.	<i>Medical Journal of Australia (Sydney)</i>
Med. Klin.	<i>Medizinische Klinik (Munich)</i>
Med. Lavoro	<i>Medicina del Lavoro (Milan)</i>
Med. Mschr.	<i>Medizinische Monatsschrift (Stuttgart)</i>
Med. Pharmacol. Exp.	<i>Medicina et Pharmacologia Experimentalis (Basel)</i>
Med. Sci. Law	<i>Medicine, Science and the Law (London)</i>
Med. Welt	<i>Medizinische Welt (Stuttgart)</i>
Med. Leg. J.	<i>Medico-Legal Journal (Cambridge)</i>
Minerva Anest.	<i>Minerva Anestesiologica (Turin)</i>
Minerva Medicoleg.	<i>Minerva Medicolegale (Turin)</i>
Mod. Hosp.	<i>Modern Hospital (Chicago)</i>
Molec. Pharmacol.	<i>Molecular Pharmacology (New York)</i>
Munchen. Med. Wschr.	<i>Munchener Medizinische Wochenschrift (Munich)</i>
N. Carolina Med. J.	<i>North Carolina Medical Journal (Winston-Salem)</i>
Naunyn Schmiedeberg. Arch. Pharm. Exp. Path.	<i>Naunyn-Schmiedeberg's Archiv fur Pharmakologie und Experimentelle Pathologie (Berlin)</i>
Nederl. T. Geneesk.	<i>Nederlands Tijdschrift voor Geneeskunde (Amsterdam)</i>
New Eng. J. Med.	<i>New England Journal of Medicine (Boston)</i>
New York J. Med.	<i>New York State Journal of Medicine (New York)</i>
Nord. Med.	<i>Nordisk Medicin (Stockholm)</i>
Nord. Psychiat. T.	<i>Nordisk Psykiatrisk Tidsskrift (Middelfart)</i>
Nova Scotia Med. Bull.	<i>Nova Scotia Medical Bulletin (Halifax)</i>
Orv. Hetil.	<i>Orvosi Hetilap (Budapest)</i>
Pharm. Weekbl.	<i>Pharmaceutisch Weekblad (The Hague)</i>
Pharmacol. Rev.	<i>Pharmacological Reviews (Baltimore)</i>
Physiol. Rev.	<i>Physiological Reviews (Bethesda)</i>
Pol. Tyg. Lek.	<i>Polski Tygodnik Lekarski (Warsaw)</i>
Postgrad. Med.	<i>Postgraduate Medicine (Minneapolis)</i>
Prensa Med. Argent.	<i>Prensa Medica Argentina (Buenos Aires)</i>
Presse Med.	<i>Presse Medicale (Paris)</i>
Proc. Roy. Soc. Med.	<i>Proceedings of the Royal Society of Medicine (London)</i>
Proc. Soc. Exp. Biol. Med.	<i>Proceedings of the Society for Experimental Biology and Medicine (New York)</i>
Przegl. Lek.	<i>Przegląd Lekarski (Crakow)</i>
Psychiat. Pol.	<i>Psychiatria Polska (Gdansk)</i>
Psychol. Rep.	<i>Psychological Reports (Missoula)</i>
Psychosom. Med.	<i>Psychosomatic Medicine (New York)</i>
Public Health Rep.	<i>Public Health Reports (Washington)</i>
Quart. J. Stud. Alcohol	<i>Quarterly Journal of Studies on Alcohol (New Brunswick)</i>
Rev. Franc. Etud. Clin. Biol.	<i>Revue Francaise d'Etudes Cliniques et Biologiques (Paris)</i>
Rev. Med. Suisse Rom.	<i>Revue Medicale de la Suisse Romande (Lausanne)</i>
Riv. Crit. Clin. Med.	<i>Rivista Critica di Clinica Medica (Florence)</i>
Roczn. Akad. Med. Marchlewski.	<i>Roczniki Akademii Medycznej imienia Juliana Marchlewskiego w Białymstoku (Bialystok)</i>
S. Afr. J. Med. Sci.	<i>South Africa Journal of Medical Sciences (Johannesburg)</i>
S. Afr. Med. J.	<i>South African Medical Journal (Cape Town)</i>

Scand. J. Clin. Lab. Invest.	<i>Scandinavian Journal of Clinical and Laboratory Investigation (Oslo)</i>
Scand. J. Gastroent.	<i>Scandinavian Journal of Gastroenterology (Oslo)</i>
Schweiz. Med. Wschr.	<i>Schweizerische Medizinische Wochenschrift (Basel)</i>
Sem. Hop. Paris	<i>Semaine des Hopitaux de Paris (Paris)</i>
Sist. Nerv.	<i>Sistema Nervoso (Milan)</i>
Southern Med. J.	<i>Southern Medical Journal (Birmingham)</i>
Surg. Forum	<i>Surgical Forum; Clinical Congress of the American College of Surgeons (Chicago)</i>
T. Norsk. Laegeforen.	<i>Tidsskrift for den Norske Laegeforening (Oslo)</i>
Texas Rep. Biol. Med.	<i>Texas Reports on Biology and Medicine (Galveston)</i>
Ther. Gegenw.	<i>Therapie der Gegenwart (Berlin)</i>
Thromb. Diath. Haemorrh.	<i>Thrombosis et Biathesis Haemorrhagica (Stuttgart)</i>
Toxic. Appl. Pharmacol.	<i>Toxicology and Applied Pharmacology (New York)</i>
Trans. Ass. Amer. Physicians	<i>Transactions of the Association of American Physicians (Philadelphia)</i>
Ugeskr. Laeg.	<i>Ugeskrift for Laeger (Copenhagen)</i>
Un. Med. Canada	<i>Union Medicale du Canada (Montreal)</i>
Virginia Med. Monthly	<i>Virginia Medical Monthly (Richmond)</i>
W. Virginia Med. J.	<i>West Virginia Medical Journal (Charleston)</i>
Wiad. Lek.	<i>Wiadomosci Lekarskie (Warsaw)</i>
Wien. Klin. Wschr.	<i>Wiener Klinische Wochenschrift (Vienna)</i>
Wien. Med. Wschr.	<i>Wiener Medizinische Wochenschrift (Vienna)</i>
Wisconsin Med. J.	<i>Wisconsin Medical Journal (Madison)</i>
Z. Biol.	<i>Zeitschrift für Biologie (Berlin)</i>
Z. Exp. Angew. Psychol.	<i>Zeitschrift für Experimentelle und Angewandte Psychologie einschliesslich Experimentelle Chirurgie (Göttingen)</i>
Z. Ges. Exp. Med.	<i>Zeitschrift für die Gesamte Experimentelle Medizin (Berlin)</i>
Z. Ges. Inn. Med.	<i>Zeitschrift für die Gesamte Innere Medizin und Ihre Grenzgebiete (Leipzig)</i>
Z. Tuberk.	<i>Zeitschrift für Tuberkulose und Erkrankungen der Thoraxorgane (Leipzig)</i>
Zbl. Arbeitsmed.	<i>Zentralblatt für Arbeitsmedizin und Arbeitsschutz (Darmstadt)</i>
Zh. Nevropat. Psikhiat. Korsakov.	<i>Zhurnal Nevropatologii i Psikiatrii imeni S.S. Korsakova (Moscow)</i>

Languages of Papers Included in the Bibliography

Language	No. of Items	Percentage	Language	No. of Items	Percentage
Afrikaans	1	.07	Japanese	12	.80
Czech	9	.60	Norwegian	5	.34
Danish	6	.40	Polish	16	1.07
Dutch	5	.34	Portuguese	2	.13
English	849	56.59	Rumanian	1	.07
Finnish	2	.13	Russian	21	1.40
French	118	7.87	Serbo-Croatian	2	.13
German	376	25.06	Spanish	19	1.27
Hungarian	4	.27	Swedish	17	1.13
Italian	34	2.26	Turkish	1	.07

Total = 1500

Years of Publication of Papers Included in the Bibliography

Period	No. of Items	Percentage	Period	No. of Items	Percentage
1899 and earlier	73	4.87	1950 to 1959	281	18.74
1900 to 1939	197	13.13	1960 to 1964	314	20.94
1940 to 1949	148	9.86	1965 and later	487	32.46

Total = 1500

Bibliography

1. Abbott, G. A., and Miller, M. J.
CARBON TETRACHLORIDE POISONING: A REPORT ON TEN CASES AT THE U.S.
MARINE HOSPITAL, SEATTLE, WASHINGTON, SINCE 1937.
Public Health Rep. (Washington), 63(2): 1619-1624 (3 ref.), 1948.
E – SEC – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – humans – acid-base,
blood pH, elect. – cardiovasc. – CNS – G.I. tract – liver, kidney – metab. proc. – respir. –
anti-infectants – *CAAAL-0 A-1327.

In a review of 10 case histories of carbon tetrachloride poisoning, alcohol seemed to be a predisposing factor in 8 cases. In 3 of the 4 fatal cases, the patient was a heavy drinker, and, in the fourth fatality, there was a history of mild alcoholism. The general clinical picture for carbon tetrachloride poisoning is: headache, nausea, vomiting, hematemesis, hematuria, icterus, oliguria, and retention of urine. Anuria and oliguria are often the primary physiological disturbances, and the development of pulmonary edema is often the immediate cause of death. At autopsy, severe kidney and liver damage were consistently found. In the 4 fatalities, the blood urea nitrogen was between 140-200 mg%, and death occurred from 3-5 days after admission to hospital. The case histories indicate that toxic reactions to carbon tetrachloride may result from either acute or chronic exposure, whereas the predisposing influence of alcohol is largely attributed to a history of chronic ingestion of large quantities of alcohol.

2. Abel, J. J.
A CRITICAL REVIEW OF THE PHARMACOLOGICAL ACTION OF ETHYL
ALCOHOL.
In: Billings, John S., ed. *Physiological Aspects of the Liquor Problem. II.* Boston: Houghton Mifflin,
pp. 1-167 (2 ref.), 1903.
E – exp. comp. – congen. stud. – humans – mammals – other org. – dose resp. – CNS – respir. –
alcohols – anesthetics – *CAAAL-0 A-1242.

The relative toxicities of the various constituents of alcoholic beverages, as compared with that of ethyl alcohol alone, are discussed. In experiments on rabbits, it was found that the addition to ethyl alcohol of 1% of the next 3 higher alcohols produced a minimal effect in increasing toxicity. Addition of 2% of 1 of the higher alcohols produced an appreciable increase in toxicity, and the addition of 4% of amyl alcohol caused such a considerable increase in toxicity that a severe type of poisoning resulted (the toxic equivalent being 4.66 g/kg for the mixture, compared to 7.44 g for ethyl alcohol). Experiments on rabbits have indicated that the esters produced in the fermentation process, such as ethyl esters of acetic, butyric, and oenanthic acids, contribute to certain alcoholic beverages, such as wines of high flavour, a more stimulating effect on respiration. The liquor known as absinthe contains 47-80% ethanol, and is highly flavoured with aromatic constituents of wormwood, anise, fennel, coriander, calamus aromaticus, hyssop, marjoram, etc. Unlike pure alcohol, which requires time to produce delirium, absinthe gives rise to hallucinations, epileptic attacks, and states of delirium almost immediately. The difference in action is ascribed to the volatile and aromatic constituents which accompany the alcohol.

3. Abel, J. J.
PHARMACOLOGICAL ACTION OF THE NON-ALCOHOLIC CONSTITUENTS OF
ALCOHOLIC BEVERAGES.
Science (Washington), 33(854): 725-727 (2 ref.), 1911.
E – general – congen. stud. – humans – mammals – CNS – respir. – *CAAAL-0 A-0470.

In a letter to the editor, the author reaffirms his position that if the alcohol were removed from highly flavoured liquor, its excessive consumption would still harm the nervous system. The aromatic

constituents of wormwood, anise, fennel, coriander, calamus, etc., would have this effect. Also, highly flavoured wines, brandy, etc., which contain larger amounts of stimulating esters, have a more pronounced effect than pure ethanol.

4. Abreu, B. E., and Emerson, G. A.
SUSCEPTIBILITY TO ETHER ANESTHESIA OF MICE HABITUATED TO ALCOHOL, MORPHIN [S/C] OR COCAIN [S/C].
Anesth. Analg. (Cleveland), 18: 294-300 (10 ref.), 1939.
 E – exp. cont. – DC (decrease) – mammals – chronic admin. – in vivo – G.I. tract – liver, kidney – metab. proc. – respir. – skel., muscle, skin – anesthetics – *CAAAL-524-D2 A-0471.

One hundred and twenty mice were exposed to 2.2 m moles/l of ether vapor in oxygen. After recovery from anesthesia, 4 groups of 30 mice each received ip one of the following: ethanol, morphine, cocaine, and saline sol. Anesthesia was given every week for 6 weeks. For the first 3 weeks, a large increase in time of induction was observed in mice treated with ethanol. After 5 weeks of treatment, a marked decrease in induction time occurred. Increase in induction time in all groups is explained as a result of hydration. Detailed tables are presented.

5. Abshagen, U., and Rietbrock, N.
ELIMINATION VON 2-PROPANOL UND IHRE BEEINFLUSSUNG DURCH ALIPHATISCHE ALKOHOLE. [Elimination of 2-propanol and the influence of aliphatic alcohols].
Naunyn Schmiedeberg Arch. Pharm. Exp. Path. (Berlin), 264(3): 212-213 (0 ref.), 1969.
 G – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vitro – blood lev. – metab. proc. – alcohols – *CAAAL-0 B-0903.

In 1 experiment, rats and dogs received 1 g/kg 2-propanol. The concentration of acetone and the initial 2-propanol concentration, as well as time and elimination constants, were tabulated. In a second experiment, the influence of methanol, ethanol, 1-propanol, and tertiary butanol on the in vitro elimination of 2-propanol, and vice versa, was investigated in rats. It was found that the elimination of 2-propanol follows an exponential function, and is different from that of ethanol and methanol, which follow linear and second-degree exponential functions, respectively. Methanol and tertiary butanol, which are not converted by alcohol dehydrogenase (ADH), did not influence 2-propanol elimination. However, ethanol and 1-propanol, which are converted by ADH, inhibited 2-propanol elimination. Conversely, 2-propanol had no inhibiting effect on 1-propanol elimination, while it significantly delayed ethanol elimination, changing the linear course of elimination of the ethanol to exponential; the former phenomenon is explained by the high affinity of 1-propanol to ADH, and by the relatively low 2-propanol concentration. It is concluded that, if the results are applicable to in vivo conditions, then 2-propanol is metabolized by ADH.

6. Adams, F. J.
A PLEA FOR THE FURTHER USE OF CARBOLIC ACID.
New York Medical Journal (New York), 70: 780-781 (0 ref.), 1899.
 E – general – case hist. – DC (antidotal) – humans – skel., muscle, skin – anesthetics – *CAAAL-0 A-0472.

The author makes a plea for the use of pure carbolic acid as a bactericide on the grounds that alcohol is a complete antidote for it. The carbolic acid-alcohol treatment has been used by him in any and all inflammatory conditions where streptococci and staphylococci are present. In addition, the history of an attempted suicide with carbolic acid is given. One pint of alcohol was administered po immediately, followed by 8 further doses of several ounces of whiskey or alcohol at various intervals for approximately three hr. The patient showed no signs of alcoholism and very little shock.

7. Adams, W. L.
THE TOXICITY OF CHLORAL ALCOHOLATE.
J. Pharmacol. Exp. Ther. (Baltimore), 69: 273-274 (0 ref.), 1940.
E – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – sed., hypnot. – *CAAAL-0
A-0473.

Injunctions against prescribing chloral hydrate in alcoholic vehicles because of the belief that chloral alcoholate is formed and constitutes the chief toxic agent of “knock-out drops”, are not substantiated in this study. The comparative hypnotic and toxic actions of chloral alcoholate and chloral hydrate in rats were determined. The results from more than 500 administrations (by stomach tube) to 162 rats show that the alcoholate is slightly less hypnotic and less toxic than the hydrate. Further studies will determine the effect of repeated administration of these compounds in hypnotic doses.

8. Adams, W. L.
THE COMPARATIVE TOXICITY OF CHLORAL ALCOHOLATE AND CHLORAL
HYDRATE.
J. Pharmacol. Exp. Ther. (Baltimore), 78(4): 340-345 (12 ref.), 1943.
E – exp. cont. – exp. comp. – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo
– dose resp. – CNS – species or sex diff. – sed., hypnot. – *CAAAL-3911-D2 A-0474.

The hypnotic potencies, acute toxicities, and chronic toxicities of sol of chloral hydrate and chloral alcoholate (made by dissolving 4 g crystalline chloral alcoholate or chloral hydrate in 100 cc distilled water) were compared in rats. Acute toxicity was also tested in rabbits, cats, and dogs. Quantitatively, in all species the depressant action of chloral alcoholate was slightly weaker than that of chloral hydrate. Thus, the widespread impression that sol containing alcohol and chloral hydrate are particularly depressant because of the formation of chloral alcoholate as a particularly toxic compound, is not substantiated.

9. Adriani, J., and Morton, R. C.
DRUG DEPENDENCE: IMPORTANT CONSIDERATIONS FROM THE
ANESTHESIOLOGIST'S VIEWPOINT.
Anesthesia and Analgesia; Current Researches (Cleveland), 47(5): 472-481 (30 ref.), 1968.
E – SEC – general – cross-tol. – drug-dep. humans – mot. perform. – CNS – alcohols – anesthetics
– barbiturates – sed., hypnot. – *CAAAL-0 B-0904.

Several types of drug dependence, as well as tolerance and withdrawal symptoms, are discussed from the anesthesiologist's point of view. The author states that difficulties occur during inhalational anesthesia or basal narcosis of chronic alcoholics, due to marked tolerance to substances such as chloral hydrate, paraldehyde, tribromethanol, and, in some cases, barbiturates. Also, cross-tolerance to ether, vinyl ether, nitrous oxide, and cyclopropane—or newer anesthetic agents, such as halothane and methoxyflurane—is well recognized. In such cases, the authors prefer the regional method of anesthesia over the general one. Dependence on chloral, paraldehyde, ethchlorvynol, and other higher molecular weight alcohols is occasionally observed in patients who have been alcoholics, and, since there is a clinical similarity in the withdrawal symptoms, as well as in response to treatment of such symptoms, it is concluded that there is a basic cellular response to these drugs.

10. Agner, K., and Belfrage, K. E.
A SPECIFIC MICRO-METHOD FOR COLORIMETRIC DETERMINATION OF
METHANOL IN BLOOD.
Acta Physiol. Scand. (Stockholm), 13: 87-94 (17 ref.), 1947.
E – exp. comp. – DC (decrease) – mammals – in vivo – blood lev. – metab. proc. – alcohols –
*CAAAL-4866-A2 A-0475.

A method for colorimetric determination of methanol is described and the rate of elimination of methanol from the blood determined. Ethanol (1.7 g/kg) and methanol (1.7 g/kg) were simultaneously injected into rabbits, after which the methanol concentration was determined at various intervals. During the first 6-7 hr, the methanol concentration was practically constant; thereafter, it followed a nearly normal pattern of decrease. It is concluded that the oxidation of methanol in the organism was prevented by the simultaneous administration of ethanol.

11. Agner, K., Höök, O., and Porat, B. von
ETANOLEFFEKTEN VID METANOLFÖRGIFTNING. [Effect of ethanol in methanol poisoning].
Svenska Läkartidningen (Stockholm), 45(21): 995-999 (3 ref.), 1948.
S – general – case hist. – DC (antidotal) – drug-dep. humans – acid-base, blood pH, elect. – metab. proc. – alcohols – *CAAAL-0 A-0476.

When ethanol is administered simultaneously with methanol, the methanol is oxidized more slowly. In methanol poisoning, measures against acidosis should be combined with ethanol administration in doses sufficient to produce approximately a 0.1% blood ethanol level. The ethanol can be given po or iv in saline or bicarbonate sol. Two cases are presented in which this treatment was used—one patient died and the other recovered; the latter received 60 ml ethanol initially and 10-20 ml po/hr to a total of 630 ml. This study is published in expanded form, in English, in Quart. J. Stud. Alcohol (New Haven), 9(4): 515-522, 1949.

12. Agner, K., Höök, O., and Porat, B. von
THE TREATMENT OF METHANOL POISONING WITH ETHANOL: WITH REPORT OF TWO CASES.
Quart. J. Stud. Alcohol (New Haven), 9(4): 515-522 (12 ref.), 1949.
E – general – case hist. – DC (antidotal) – drug-dep. humans – blood lev. – acid-base, blood pH, elect. – metab. proc. – respir. – senses – alcohols – *CAAAL-4359-N8 A-0477.

This article is an expanded version of that published in Swedish in Svenska Läkartidningen (Stockholm), 45(21): 995-999, 1948. Two cases of methanol poisoning are reported. In both cases the patients were treated with ethanol and bicarbonate. The ethanol retarded the oxidation of methanol, thereby lowering or preventing the concentration of toxic products arising from methanol oxidation. Amounts of ethanol sufficient to maintain a blood ethanol level of 0.1% were needed to prevent the appearance of methanol metabolites. The rate of methanol oxidation was checked constantly and ethanol administered accordingly. In one case the symptoms of methanol poisoning disappeared and the patient recovered. The other one died.

13. Agner, K., Hellström, R., Nilsson, C. G., and Reichard, H.
BEHANDLING AV TRÄSPRITSFÖRGIFTNING MED ETYLALKOHOL OCH PERITONEAL DIALYS. [Treatment of methanol intoxication with ethanol and peritoneal dialysis].
Opuscula Medica (Stockholm), 8: 183-186 (7 ref.), 1963.
S – general – case hist. – DC (antidotal) – humans – blood lev. – acid-base, blood pH, elect. – metab. proc. – alcohols – *CAAAL-0 A-1364.

A 27 yr-old man was hospitalized 12 hr after having drunk methanol (following the fatal poisonings of 2 companions). Outward symptoms of poisoning were absent. The blood methanol concentration was 3.5°/oo, and the standard bicarbonate level 14 meqv/l. During the first 12 hr, a total of 140 g 40% ethanol and 35 g bicarbonate were administered; after that, 50 g ethanol and standard bicarbonate were given. Since the blood methanol level was extremely high, peritoneal dialysis was performed. During the first 36 hr of dialysis, 33 g methanol were eliminated in the first 12 hr, 24 g during the

second 12 hr, and 17 g during the third 12 hr. During dialysis, the patient felt well—he ate and drank, voided normal quantities of urine, and had normal serum electrolytes. Dialysis and ethanol were stopped when blood methanol reached 0.4°/oo. Over a 3-day period, a total of 3 l of ethanol was administered. 3 days after the end of dialysis, the patient was discharged as fit. In conclusion, the importance of administration of ethanol in adequate amounts, and of correction of acidosis, is emphasized. Peritoneal dialysis in the simple and, for the patient, painless way described can eliminate large amounts of methanol. “Accelerated diuresis” with the help of iv mannitol is recommended for less severe cases of poisoning.

14. Ahlquist, R. P.

EXPERIMENTAL ALCOHOL TOLERANCE AND THE REACTIONS OF ALCOHOL-TOLERANT ANIMALS TO OTHER DRUGS.

Ph.D. Thesis, University of Washington, Seattle, Wash., U.S.A., 61 pp. (40 ref.), 1940.
E – exp. cont. – cross-tol. – mammals – chronic admin. – in vivo – blood lev. – other drug lev. – CNS – anesthetics – barbiturates – *CAAAL-0 A-1328.

Alcohol tolerance was induced in adult, male, albino rabbits, weighing from 1500 to 3500 g, by the daily ip injection for 25 days of a 20% alcohol sol in doses of 1.5 g/kg. In controlled experiments, the effect of induced alcohol tolerance on the action of various drugs was then tested. The drugs were administered 48 hr after the last alcohol injection, to ensure complete elimination of alcohol from the tissues, thereby preventing the possibility of a combined reaction of alcohol and the drugs. 2 hypnotic drugs, pentobarbital sodium (20 mg/kg, 2% sol in distilled water, iv) and evipal (40 mg/kg, 4% aqueous sol iv) showed a 40% decrease in the sleeping time produced by an iv dose of alcohol. The ether required to abolish the righting reflex was increased by about 45% in the alcohol-tolerant rabbits, and a greatly-exaggerated excitement stage in the induction of anaesthesia was produced. It is concluded, therefore, that alcohol-tolerant animals are much more resistant to the depressant effects of barbiturates and ether. No effect on amidopyrine analgesia or metrazol stimulation was produced.

15. Ahlquist, R. P., and Dille, J. M.

REACTIONS OF ALCOHOL TOLERANT RABBITS TO PENTOBARBITAL, EVIPAL, ETHER, AMIDOPYRINE AND METRAZOL.

J. Pharmacol. Exp. Ther. (Baltimore), 70: 301-308 (7 ref.), 1940.
E – exp. cont. – exp. comp. – cross-tol. – mammals – chronic admin. – in vivo – CNS – analg., antipyret. – anesthetics – barbiturates – stimulants – *CAAAL-2572-D2 A-0478.

Tolerance to alcohol, as measured by a decreasing response, can be easily produced in rabbits by the daily ip administration of 1.5 g of alcohol/kg for 15 to 25 days. Alcohol-tolerant rabbits are much more resistant than normal animals to the depressant effects of pentobarbital, evipal and ether. Cross-tolerance between alcohol and other central nervous system depressants is hypothesized.

16. Akabane, J., and Ikomi, F.

EFFECTS OF DISULFIRAM (TETRAETHYLTHIURAMDISULFIDE) AND ITS RELATED COMPOUNDS ON THE METABOLISM OF ALCOHOL.

Shinshu Daigaku, Igakubu (Shinshu University Medical Journal) (Matsumoto), 3(4): 345-351 (7 ref.), 1958.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (sensit.) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – miscellaneous – unclass. ther. agents – *CAAAL-0 A-1365.

Alcohol in 20% sol in saline was given iv at a rate of 2.0 cc/min, up to 6.25 cc/kg, to adult albino rabbits (2.0-3.5 kg). Blood was drawn from the heart at 15 min and at 1, 2, 3, and 4 hr after alcohol, and analyzed for acetaldehyde. All rabbits first received a control injection of alcohol. The alcohol administration and acetaldehyde analyses were repeated 18-20 hr after an oral dose of 0.1-0.3 g/kg

of 1 of 10 compounds related to disulfiram: disulfiram, tetramethylthiuramdisulfide (TMTD), diethyl-diphenylthiuramdisulfide (EPTD), dimorpholynylthiuramdisulfide (DMTD), tetramethylthiurammonosulfide (TMTM), sodium diethyldithiocarbamate (SDEDC), zinc diethyldithiocarbamate (SDEDC), zinc diethyldithiocarbamate (ZDEDC), dibenzothiazylidysulfide (DBTD), thiocarbanilide (TC), and mercaptobenzothiazol (MBT). Of these compounds, the most pronounced "disulfiram effect" was produced by TMTM, the blood acetaldehyde level rising to 10-15 times that of control values; alcohol intoxication symptoms were greatly intensified and prolonged. Second and third in order of effectiveness, respectively, were TMTD and disulfiram. DMTD, SDEDC, ZDEDC, and TC had an effect which was significant, but less than that of disulfiram. EPTD, DBTD, and MBT exhibited an uncertain effect or no effect. The relationship between chemical structure and the disulfiram-like action of these compounds is discussed.

17. Akabane, J., Nakanishi, S., Kohei, H., Matsumura, R., and Ogata, H.
EFFECT OF SULFONYLUREA DERIVATIVE ON THE METABOLISM OF ALCOHOL:
EXPERIMENTS WITH ISOLATED PERFUSED LIVER.
Shinshu Daigaku, Igakubu (Shinshu University Medical Journal) (Matsumoto), 8: 71-79 (12 ref.),
1963.
E – exp. cont. – DC (unchanged) – DC (sensit.) – mammals – acute admin. – chronic admin. – in
vitro – blood lev. – liver, kidney – hormones, hormone antag. – *CAAAL-0 A-1366.

The influence of tolbutamide on the rate of alcohol metabolism was determined in isolated perfused livers from male rabbits (approximately 3 kg) which had received 100 mg/kg/day tolbutamide po for more than 4 weeks; results were compared to the rate in livers of normal animals. The perfusate consisted of whole blood from animals which had received daily doses of tolbutamide, and was constantly equilibrated with a mixture of 95% oxygen and 5% carbon dioxide. Test substances were added to the perfusate in the venous reservoir. The livers were perfused for periods of 2 1/2 hr, and blood samples for analysis were drawn from the venous reservoir at regular intervals. The rate of alcohol disappearance from the blood in the perfusion of livers removed from pretreated animals was similar to that in the intact livers of normal animals, whereas perfusing blood acetaldehyde concentrations were significantly higher than in the normal liver. An increase in the blood sugar level, observed in control experiments, was less obvious after the administration of alcohol into the perfusate, and no change in the liver glycogen content was observed with or without alcohol administration. Perfusing blood lactate levels remained at the initial values after alcohol administration, whereas perfusing blood pyruvate and alpha-ketoglutarate levels fell markedly after the administration of alcohol.

18. Alajmo, B., and Accardi, V.
L'AMBLOPIA TOSSICA ALCOOLICO-TABAGICA (CONTRIBUTO SPERIMENTALE
ALLA ANATOMIA PATOLOGICA E PATHOGENESI E CONSIDERAZIONI CLINICHE).
[Toxic tobacco-alcohol amblyopia (experimental contribution to the pathological anatomy and
pathogenesis; clinical considerations)].
Boll. Oculist. (Bologna), 9: 273-295, and 361-379 (78 ref.), 1930.
I – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. – in vivo – CNS
– senses – *CAAAL-2293-G2 A-0479.

A critical review of the literature is presented and original experiments with 12 rabbits described. Alcohol was given po (15 to 30 g of 30% sol for 25 to 104 days) to 4 rabbits. 3 rabbits were compelled to inhale the smoke of 1 to 4 cigarettes every day during a period of 16 to 121 days. 5 rabbits were intoxicated with both alcohol and tobacco for 18 to 72 days. In all three groups, degenerative changes were observed in some fibers of the optic nerve and in the ganglionic cells of the posterior pole of the retina.

19. Albert, S. N., Rea, E. L., Duverney, C. A., Shea, J., and Fazekas, J. F.
THE USE OF CHLORPROMAZINE IN THE TREATMENT OF ACUTE ALCOHOLISM.
 Med. Ann. D.C. (Washington), 23(5): 245-247 (4 ref.), 1954.
 E – SEC – exp. – DC (unchanged) – drug-dep. humans – acute admin. – in vivo – cardiovasc. – CNS
 – tranquilizers – *CAAAL-6945-N10 A-0480.

Chlorpromazine was given by vein, muscle, or mouth to 21 alcoholic patients with delirium tremens and to 43 with psychomotor agitation. They fell asleep within 15 min if the drug was given iv, and within an hr if administered by other routes. Acute hypotension was observed only after the iv injection. No depression of the nervous system or synergism with alcohol was observed, and no convulsions occurred. The effects of chlorpromazine suggest that its action is on the hypothalamus.

20. Alha, A.
NS. KOLINAJOUKKOMYRKYTYS. [Mass poisoning due to hair lotion].
 Duodecim (Helsinki), 66: 659-666 (17 ref.), 1950.
 Fi – ES – SEC – DC (add., infra-add., unspec. incr.) – drug-dep. humans – anti-infectants –
 *CAAAL-5652-E4 A-0481.

32 certain cases (17 fatal, 15 non-fatal) and 10 uncertain cases (1 fatal, 9 non-fatal) were treated for carbon tetrachloride poisoning. Most were chronic alcoholics, many of whom were known abusers of alcoholic technical products; origin of the poisoning was a hair lotion containing alcohol and 1.6% carbon tetrachloride. The socio-medical, clinical, patho-anatomical, and chemical aspects are described in detail. No specific treatment was followed, due to individual variation in susceptibility. Measures attempted consisted of prophylactic protection of the organism and treatment for circulatory insufficiency and uremia. That alcohol aggravates the character of the poisoning is confirmed. The author points out that physicians are not generally aware of the possibility of this kind of poisoning. This study is published in expanded form, in English, in Ann. Med. Intern. Fenn. (Helsinki), 39(Suppl. 8): 32 pp., 1950.

21. Alha, A.
CARBON TETRACHLORIDE MASS POISONING.
 Ann. Med. Intern. Fenn. (Helsinki), 39(Suppl. 8): 32 pp. (37 ref.), 1950.
 E – SEC – DC (add., infra-add., unspec. incr.) – drug-dep. humans – anti-infectants –
 CAAAL-5652-E4 A-0482.

This article is an expanded version of that published in Finnish in Duodecim (Helsinki), 66: 659-666, 1950. 32 certain cases (17 fatal, 15 non-fatal) and 10 uncertain cases (1 fatal, 9 non-fatal) were treated for carbon tetrachloride poisoning. Most were chronic alcoholics, many of whom were known abusers of alcoholic technical products; origin of the poisoning was a hair lotion containing alcohol and 1.6% carbon tetrachloride. The socio-medical, clinical, patho-anatomical, and chemical aspects are described in detail. No specific treatment was followed, due to individual variation in susceptibility. Measures attempted consisted of prophylactic protection of the organism and treatment for circulatory insufficiency and uremia. That alcohol aggravates the character of the poisoning is confirmed. The author points out that physicians are not generally aware of the possibility of this kind of poisoning.

22. Alha, A.
CALCIUM CHLORIDE AND ALCOHOL: EFFECT OF THERAPEUTIC DOSES OF CALCIUM CHLORIDE ON THE CLINICAL STAGE OF ALCOHOLIC INTOXICATION AND ON BLOOD ALCOHOL.
 Ann. Med. Exp. Biol. Fenn. (Helsinki), 29(2): 125-136 (12 ref.), 1951.
 E – exp. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – blood lev. –
 hormones, hormone antag. – *CAAAL-5923-A1 A-0483.

Three volunteer subjects received "a measured amount" of 30.6% alcohol sol mixed with water (1 g/kg body wt) po, plus calcium chloride (CaCl_2) (2 g in 10% sol po, or 10 ml in 10% sol iv). Blood alcohol was not influenced by CaCl_2 . When alcohol was taken immediately afterwards by mouth, the symptoms were a little less pronounced than when alcohol was taken alone. Given iv, CaCl_2 had no effect 1 hr after alcohol, but seemed to reduce the degree of intoxication 2 to 3 hr after alcohol. Any reduction in the degree of intoxication produced by CaCl_2 may be due to its effect on neural functions.

23. Alha, A., and Isotalo, A.

LISÄTEKIJOISTÄ KUOLEMAAN JOHTANEISSA LÄÄKEAINEMYRKYTYKSISSÄ.

[Contributory factors in fatal cases of drug poisoning].

Suomen Lääkärilehti (Helsinki), 19: 143-151 (2 ref.),

1964.

Fi – SEC – case hist. – DC (unspec.) – med.-leg. – humans – blood lev. – alcohols – analg., antipyret. – autocoids – barbiturates – *CAAAL-0 A-0484.

This article is a shorter version of the one appearing in English by the same authors (Ann. Med. Exp. Biol. Fenn. (Helsinki), 42(Suppl. 1): 31 pp., 1964). Described and discussed are 458 cases of fatal poisoning. The kind of examinations diagnosing the cause of death and the reliability of the diagnosis were investigated from the viewpoint of the chemical examination, the autopsy, the certification of death, etc. Different aspects of the evaluation of deaths from acute poisoning were considered. Special features of mortality from poisonings were the frequency of suicides with hypnotics; combinations of drugs, a drug plus alcohol, or parathion; the great number of accidental alcoholic poisonings; and the frequency of child poisonings.

24. Alha, A., and Isotalo, A.

ON ACUTE FATAL POISONINGS IN FINLAND IN 1958 AND THEIR DETECTION: A STUDY ON THE CAUSES OF DEATH, CLASSIFICATION OF DEATHS, DIAGNOSTIC BASIS, ADDITIONAL FACTORS, BACKGROUND AND STATISTICAL CLASSIFICATION OF ACUTE FATAL POISONINGS REPORTED IN THE OFFICIAL CAUSE OF DEATH STATISTICS.

Ann. Med. Exp. Biol. Fenn. (Helsinki), 42(Suppl. 1): 31 pp. (17 ref.),

1964.

E – SEC – case hist. – DC (unspec.) – med.-leg. – humans – blood lev. – alcohols – analg., antipyret. – autocoids – barbiturates – *CAAAL-0 A-0485.

This article is an expanded version of the one appearing in Finnish by the same authors (Suomen Lääkärilehti (Helsinki), 19: 143-151, 1964). Described and discussed are 458 cases of fatal poisoning. The kind of examinations diagnosing the cause of death and the reliability of the diagnosis were investigated from the viewpoint of the chemical examination, the autopsy, the certification of death, etc. Different aspects of the evaluation of deaths from acute poisoning were considered. Special features of mortality from poisonings were the frequency of suicides with hypnotics; combinations of drugs, a drug plus alcohol, or parathion; the great number of accidental alcoholic poisonings; and the frequency of child poisonings. The different aspects are discussed in considerable detail.

25. Allegri, A.

COCAINA, ALCOOL, DINITROFENOLO E BLU DI METILENE NELLA INTOSSICAZIONE SPERIMENTALE DA BARBITURICI. [Cocaine, alcohol, dinitrophenol and methylene blue in experimental barbiturate intoxication].

Boll. Soc. Ital. Biol. Sper. (Naples), 10: 48-51 (15 ref.),

1935.

I – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – barbiturates – *CAAAL-0 A-0486.

The literature (up to 1934) on experimental treatment of barbiturate intoxication is reviewed. The effects of cocaine treatment of barbiturate poisoning in guinea pigs, rats, and 1 dog; of iv administra-

tion of alcohol in guinea pigs, dogs, and rabbits; of dinitrophenol in guinea pigs and rats; and of methylene blue in guinea pigs and pigeons, are discussed. Alcohol seemed only to be effective in rabbits which had received doses of gardenal just over the lethal minimum (0.10-0.15 g/kg). In dogs with high lethal doses of gardenal, in guinea pigs with lethal doses of veronal, and in one rabbit with veronal, alcohol failed to arrest the progress of poisoning. Allegri, therefore, only partially confirms the results of Carrière, G., Huriez, Cl., and Willoquet, P., (C.R. Soc. Biol. (Paris), 116: 188-190, 1934) which purported that phenobarbital and alcohol are antagonistic.

26. Allison, B. R.

THE TREATMENT OF ACUTE CARBON TETRACHLORIDE POISONING WITH A REPORT OF TWO CASES.

Ann. Intern. Med. (Philadelphia), 16: 81-93 (20 ref.),

1942.

E – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – drug-dep. humans – blood comp., sites, lymph – cardiovasc. – liver, kidney – respir. – anti-infectants – *CAAAL-0 A-0487.

Two cases of acute carbon tetrachloride poisoning are presented. The first, that of a 9 yr-old boy who received a large dose by inhalation, showed evidence of liver and kidney damage, followed by slow convalescence. The second concerned a 48 yr-old man who died 10 days after drinking cleaning fluid. The man was an alcoholic, and had been imbibing before he drank the fatal potion, thinking it to be whiskey; according to the author, the CCl_4 toxicity was undoubtedly increased by alcohol.

27. Alstott, R. L., Brown, D. J., and Forney, R. B.

ENZYMATIC ACTIVITY PATTERNS IN THE RABBIT AFTER CAFFEINE-ETHANOL ADMINISTRATION.

Toxic. Appl. Pharmacol. (New York), 17(1): 296 (0 ref.),

1970.

E – abst. – exp. cont. – DC (unspec.) – mammals – acute admin. – in vivo – blood lev. – psychol. perform. – CNS – metab. proc. – stimulants – *CAAAL-0 B-0905.

Although caffeine has been found to be an antagonist of alcohol, it has also been revealed that caffeine administration fails to alleviate alcohol-induced psychomotor impairment; moreover, enhancement of depression and toxicity has been shown in rats after combined caffeine-alcohol administration. To attempt to resolve these conflicting findings, the interaction of caffeine and alcohol was studied in Dutch rabbits. Depression was measured by a shock avoidance box. Urea nitrogen, glucose, and drug levels were measured in blood samples taken at intervals over a 5-hr period. Blood drawn by cardiac puncture 24 hr after administration of either drug alone or both drugs combined was analyzed for serum GOT, GPT, LDH, and isozymes, amylase, alkaline phosphatase, and urea nitrogen. It was found that the administration of 100 mg/kg caffeine, with or without 1.0 g/kg ethanol, produced changes from control values in some of the tests. The results of the enzyme studies and urea nitrogen determinations were evaluated in order to locate organ system changes, and the other parameters were studied to obtain information on possible metabolic alterations.

28. Alstott, R. L., and Forney, R. B.

PERFORMANCE STUDIES IN RABBITS, RATS AND MICE AFTER ADMINISTRATION OF CAFFEINE OR L-METHYLXANTHINE, SINGLY AND WITH ETHANOL.

Fed. Proc. (Bethesda), 30(2): 568Abs (1 ref.),

1971.

E – abst. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – mot. perform. – psychol. perform. – CNS – stimulants – *CAAAL-0 B-0906.

Performance studies were conducted on rats and rabbits, using a shock avoidance apparatus, to study the antagonistic effect of caffeine (CAF) and l-methylxanthine (l-MX) on ethanol depression. In addition, sleeping time studies were performed on mice. Rats received 100 mg/kg of CAF and 1.0 g/kg ethanol, and rabbits received the same CAF dose plus 2.0 g/kg ethanol. l-MX, the major

metabolite of CAF, was given only to rats in a dose of 50 mg/kg, with or without 2 g/kg ethanol. The gross depression in rats and rabbits was significantly greater after CAF plus ethanol than after ethanol alone. After l-MX, with or without ethanol, the animals were alert, but were physically unable to react in their trained manner. From the results, confirmed by sleeping time experiments with mice, the author concludes that CAF does not antagonize ethanol depression, and that the observed impairment, as shown by the shock avoidance apparatus, might be motor, rather than total, impairment.

29. Alves, M. A. M.
 ACÇÕES ORGANOTRÓPICAS DAS SULFONAMIDAS (CONTRIBUIÇÃO PARA O SEU ESTUDO). [Organotropic action of sulfonamides (a contribution to their study)].
 Arq. Pat. (Lisbon), 18(2): 81-250 (561 ref.), 1946.
 P – FS – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo
 – anti-infectants – *CAAAL-0 A-0488.

A study was made on the effects of alcohol and sulfonamides administered individually and in combination to white mice. In two experiments, using 30 white mice of both sexes, sulfonamide was administered into the stomach in a dose of 0.5 mg/g, followed, five hr later, by alcohol (7.8-10 cc 10% alcohol sc) in a serum given sc. The results (tabulated) indicate, that mortality ensued in 100% of the animals treated with both alcohol and sulfonamide, and that only 33% (2 out of 6) of the animals injected with alcohol alone died. All animals (3) receiving sulfonamide alone survived.

30. Amado Ledo, E.
 TRATAMIENTO DE LA INTOXICACION ALCOHOLICA. [treatment of alcohol intoxication].
 Archivos del Hospital Universitario (Havana), 8: 262-265 (8 ref.), 1956.
 Sp – general – DC (antidotal) – humans – CNS – G.I. tract – respir. – anti-infectants – hormones, hormone antag. – nutritive agents – sed., hypnot. – stimulants – tranquilizers – unclass. ther. agents
 – *CAAAL-7862-M3 A-0489.

An outline of treatment of alcoholism is presented. For acute intoxication, glucose, sedatives, cerebral stimulants, penicillin, vitamins, etc., are recommended. Complete alcohol abstinence from the start of treatment is considered necessary, and if it cannot be maintained on an out-patient basis, then hospitalization is recommended. Psychotherapy, disulfiram, and hormones (adrenal) are also discussed.

31. Amagat
 RECHERCHES EXPÉRIMENTALES SUR L'ANTAGONISME EN THÉRAPEUTIQUE—ANTAGONISME DE L'ALCOOL ET DE LA STRYCHNINE.
 [Experimental research on antagonism in therapy—antagonism of alcohol and strychnine].
 Journal de Thérapeutique (Paris), 3: 378-383 (0 ref.), 1876.
 F – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – CNS – stimulants
 – *CAAAL-0 A-0490.

Of two rabbits injected with 1 mg of strychnine sulphate, one was given 12 g of alcohol 2 min later and recovered, the other was given 10 g of alcohol 5 min later, suffered convulsions, and died. A third rabbit was given 1 1/2 mg of strychnine sulphate, and 5 min later, 15 g of alcohol. It did not suffer convulsions, but died from the alcohol. It is concluded that a toxic dose of strychnine is neutralized by a non-toxic dose of alcohol, provided that the quantity of strychnine does not exceed certain limits.

32. American Medical Association, Committee on Alcoholism and Addiction
DEPENDENCE ON BARBITURATES AND OTHER SEDATIVE DRUGS.
J.A.M.A. (Chicago), 193(8): 107-111 (0 ref.), 1965.
E – SEC – general – conj. addict. – DC (add., infra-add., unspec. incr.) – humans – CNS – barbiturates
– *CAAAL-0 B-0500.

The Committee on Alcoholism of the American Medical Association has prepared a statement on sedative addiction. The historical development of drug abuse, the definition of the problem—barbiturate production, medical need, and sedative use, misuse, and abuse—, psychiatric considerations, patterns of abuse—types of abusers, suicide—, diagnosis of misuse and abuse—general intoxication, tolerance, withdrawal syndrome—, treatment of the withdrawal syndrome, and definitive treatment and aftercare are discussed, and an appendix of barbiturates and drugs with barbiturate-like action on the U.S. market is added. Of the 4 main types of abusers, 1 category includes conjunctive addiction to barbiturates and other drugs, primarily alcohol and/or opiates. It is stated that, "Many alcoholics attempt to counteract the withdrawal effects of alcohol with barbiturates. Frequently, alcohol and barbiturates are combined in an attempt to obtain effects that surpass those of either. This practice is especially hazardous, as the cumulative effects can easily result in very serious intoxication or death."

33. American Medical Association, Council on Drugs
REEVALUATION OF TRANLYCYPROMINE SULFATE (PARNATE SULFATE).
J.A.M.A. (Chicago), 189(10): 763-764 (3 ref.), 1964.
E – SEC – general – DC (add., infra-add., unspec. incr.) – humans – enzymes – *CAAAL-0
A-0491.

The actions and uses of tranlycypromine sulfate are discussed. Its effectiveness is compared to other antidepressant agents; its indications and contraindications, adverse reactions, precautions, and optimal dosage are described. "While undergoing therapy with tranlycypromine or other monoamine oxidase inhibitors, patients should be warned not to eat cheese or to take proprietary drugs that contain pressor and antihistaminic agents (e.g., certain cold remedies, hay fever preparations, or anorexiant) or drink alcoholic beverages."

34. Ammon, H. P. T.
ALKOHOL UND PUROMYCIN C, ZWEI SICH VERSTÄRKENDE FAKTOREN BEI DER
ENTSTEHUNG DER FETTLIEBER. [Alcohol and puromycin C, two augmenting factors in the
pathogenesis of fatty liver].
Z. Ges. Exp. Med. (Berlin), 152: 56-61 (13 ref.), 1970.
G – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – absorp.,
distrib., stor. – glands – liver, kidney – *CAAAL-0 B-0907.

An experiment was conducted to determine whether or not the deposition of fat in the liver, influenced by a puromycin C-induced impairment of protein synthesis, is increased by the addition of alcohol. Mice were divided into 4 groups. Group 1 received .10 µg/g puromycin C ip 10 times at 90 min intervals. Group 2 received a single injection of 4 mg/g alcohol iv (20% w/v), group 3 received puromycin and alcohol in combination in the above dosages, and group 4 served as control. In each group the glycerol-1-P concentration in liver, and the total fatty acids in liver and epididymis adipose tissue were determined. It was found that there was no significant change in glycerol-1-P in liver in group 1, and an increase in groups 2 and 3. With respect to total fatty acid concentration in epididymal tissue, there was no significant change in group 1, and a decrease in groups 2 and 3. Total fatty acids increased in groups 1 and 2, and, in group 3, there was an increase beyond the levels of the puromycin-only group. It is concluded that the decrease in total fatty acids in epididymal adipose tissue and the increase of glycerol-1-P in the liver induced by administration of alcohol alone can also

be detected in animals in which protein synthesis is impaired by puromycin C. Factors increasing the liver lipid content by different mechanisms may be additive in their effect.

35. Amorosi, O.
INTOSSICAZIONE ALCOOLICA E NARCOSI. [Alcoholic intoxication and narcosis].
Rinascenza Medica (Naples), 8: 151-153 (0 ref.), 1931.
I – ES – FS – GS – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. –
in vivo – liver, kidney – metab. proc. – respir. – skel., muscle, skin – anesthetics – *CAAAL-0
A-1367.

The influence of ether and chlorform narcosis on cellular respiration after alcohol intoxication was investigated in guinea pigs. 2 normal and 2 alcohol-intoxicated animals were placed in a controlled environment into which ether or chloroform gas-vapour were introduced for a period of 1-2 hr until death. Comparative tissue activity in sections of brain, lung, liver, kidney, and muscle was determined by the dehydrogenation method of Lipschitz. The intoxicated animals manifested a more violent and longer-lasting initial period of excitement, the excitement reappearing at intervals during the narcosis; the chloroform-induced excitement was of a minor duration compared to that induced by ether. To produce death in the intoxicated animals, it was necessary only to slightly increase the quantity of narcotic, whereas, in the normal animals, a triple quantity of narcotic was required. Regarding narcotic-induced tissue alterations, a greater inhibition of the process of dehydrogenation was found in tissue of intoxicated animals, as compared to controls. The diminished respiration in tissue of intoxicated animals extended to all tissue types, the respiratory quotient of the brain, liver, and kidney being more decreased by chloroform than by ether. Conversely, the lung quotient was more diminished by ether than by chloroform. The respiratory quotient of muscle tissue was increased by chloroform, and slightly decreased by ether.

36. Amsel, L. P., and Levy, G.
EFFECT OF ETHANOL ON THE CONJUGATION OF BENZOATE AND SALICYLATE
WITH GLYCINE IN MAN.
Proc. Soc. Exp. Biol. Med. (New York), 135(3): 813-816 (6 ref.), 1970.
E – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – acute admin.
– in vivo – other drug lev. – metab. proc. – analg., antipyret. – *CAAAL-0 B-0908.

Healthy young male volunteers were given 2-5 g benzoic acid as sodium benzoate po. Alcohol (50 ml) was administered po 15 min before or 90 min after benzoate. Urine was collected at intervals and analyzed for benzoate, hippurate, and benzoic glucuronide. In another experiment, 2 g of salicylic acid were administered po, followed 2 hr later by 50 ml ethanol po. Some subjects instead received 1 g salicylic acid po, followed 3 hr later by 3.2 g benzoic acid as sodium benzoate po; 50 ml ethanol were taken po over 10 min, starting 25 min before the benzoate, and 10 ml ethanol were taken with the benzoate. Salicylate and its metabolites in urine were determined. Ethanol inhibited the formation of hippurate from benzoate, but had no effect on the maximum rate of formation of salicylurate from salicylate. This finding suggests that ethanol depresses the activity of glycine N-acylase (the enzyme which catalyzes the transfer of the activated drug to glycine), or decreases the availability of glycine to the conjugation system. This is consistent with other evidence showing that the conjugation of benzoate and salicylate with glycine in man involves different rate-limiting steps.

37. Andersen, A. H.
DØDSFALD EFTER INDGIFT AF TETRAPON OG HYPNOPEN TIL
ALKOHOLPÅVIRKET PERSON. [Death after intake of tetrapon and hypnopen in a person
under the influence of alcohol].
Ugeskr. Laeg. (Copenhagen), 116(39): 1400-1401 (1 ref.), 1954.
Da – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – blood lev. – other drug
lev. – analg., antipyret. – *CAAAL-7026-E3 A-0492.

A severely drunk, unconscious man died after a sc injection of tetrapon and hypnophen. There was 2.08°/oo alcohol in his blood and 8.63°/oo in his urine. K. Møller's warning concerning treatment of drunken patients with morphine (Ugeskr. Laeg. (Copenhagen), 114(50): 1785-1793, 1952) is repeated and confirmed.

38. Andersén, K.

RISK FÖR MEPROBAMAT-MISSBRUK HOS ALKOHOLMISSBRUKARE! [Risk of misuse of meprobamate in alcohol abusers!].

Svenska Läkartidningen (Stockholm), 56: 2356-2360 (7 ref.),

1959.

S – general – case hist – conj. addict. – DC (add., infra-add., unspec. incr.) – drug-dep. humans – tranquilizers – *CAAAL-9086-L3 A-0493.

The literature on habituation and withdrawal syndromes with respect to meprobamate is reviewed. A progressively increasing proportion of alcoholic patients began to misuse meprobamate in conjunction with alcohol at the alcoholism clinic in Stockholm between 1956 and 1958. When the drug was first used at the clinic in 1956, it was considered harmless and a maximum daily dose of 2.4 g was prescribed; however, some patients increased the dose and discovered that, when meprobamate was taken in conjunction with alcohol, the desired euphoria was induced. In 1957 there were 9 patients taking 12 g/day, and by 1958, 16 patients were found to be misusing the drug.

39. Anonymous (Any Questions?)

ALCOHOL AND ANTICOAGULANTS.

Brit. Med. J. (London), 2: 1615 (0 ref.),

1960.

E – general – DC (add., infra-add., unspec. incr.) – humans – liver, kidney – coagulants – *CAAAL-0 A-0494.

Patients on long-term anticoagulant therapy are warned against taking large amounts of alcohol in any form. Any patient who habitually consumes a lot of alcohol should certainly be advised to restrict his intake. The effects of alcohol on the liver and the well-known sensitivity of patients with liver disease to anticoagulant drugs fully justify this advice.

40. Anonymous (Any Questions?)

ALCOHOL AND BARBITURATES.

Brit. Med. J. (London), 1: 232 (2 ref.),

1953.

E – general – DC (add., infra-add., unspec. incr.) – humans – CNS – barbiturates – *CAAAL-6498-D3 A-0495.

In answer to the question, "Do alcohol and barbiturates potentiate each other?" it is stated that: "Barbiturates and alcohol are both cerebral depressants. Consequently the effect of one will certainly be potentiated by a dose of the other. Whether they are pharmacologically synergistic to one another is more controversial."

41. Anonymous (Notes and Comments)

ALCOHOL AND BARBITURATES.

Brit. Med. J. (London), 1: 846 (2 ref.),

1953.

E – general – DC (add., infra-add., unspec. incr.) – humans – barbiturates – *CAAAL-6498-D3 A-0496.

The terms synergism and potentiation are discussed. No reports on the effect of alcohol and barbiturates when used together, on ataxia, euphoria, etc., have been found, although synergism has been demonstrated experimentally between ethanol and phenobarbital by Jetter and McLean (Arch. Path.

(Chicago), 36: 112-122, 1943), and, in accidental human poisonings, Sandberg (*Acta Physiol. Scand.* (Stockholm), 22: 311-325, 1951) has demonstrated a potentiative type of synergism between ethanol and 6 short-acting barbiturates, the degree of potentiation varying with the dose and the derivative used.

42. Anonymous (Questions and Answers)
ALCOHOLIC BEVERAGES AND ORALLY GIVEN HYPOGLYCEMIC DRUGS.
J.A.M.A. (Chicago), 173: 128 (3 ref.), 1960.
E – general – DC (sensit.) – humans – cardiovasc. – CNS – respir. – skel., muscle, skin – hormones, hormone antag. – *CAAAL-9248-E3 A-1252.

A question is asked concerning a 58 yr-old male diabetic who experienced an unusual reaction to alcoholic beverages while taking orally-given hypoglycemic drugs. 5 days after being placed on chlorpropamide therapy (375 mg/day), he drank an alcoholic beverage, and, within 10 min, he experienced a flushed face, slight discomfort in the chest, and a desire to lie down. The reaction lasted for 45 min. He was changed to tolbutamide therapy, and had a similar but milder reaction after ingestion of alcohol. The author comments that intolerance to alcohol has been reported in patients receiving sulfonylurea compounds. Dolger reported 5 cases of alcohol intolerance among 500 patients receiving tolbutamide. Blöch and Lenhardt observed an even greater incidence of alcohol intolerance (11.6% of 215 patients) in diabetics receiving 0.25-0.50 g of chlorpropamide every other day. The mechanism of the reaction is unknown, and prevention consists of abstinence from either alcohol or sulfonylurea compounds.

43. Anonymous (Queries and Minor Notes)
ALCOHOLIC DRIVERS.
J.A.M.A. (Chicago), 160: 818 (0 ref.), 1956.
E – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev. – other drug lev. – autocoids – *CAAAL-7487-U27 A-0497.

Question: In 2 cases concerning motorists who had ingested alcohol in addition to an antihistaminic preparation, alcometer tests showed a legal level of intoxication, although each person claimed that less than 3 oz of whiskey had been ingested. Can the ingestion of antihistamines affect the alcohol determination as performed by the alcometer? Answer: No, the presence of these drugs has no effect on the alcohol determination of breath, blood, or other body fluids. When alcohol is taken in addition to the antihistamines, the symptoms of intoxication are more evident.

44. Anonymous (News and Notes. Medico-Legal)
ANTI-HISTAMINES AND ALCOHOL.
Brit. Med. J. (London), 1: 403 (0 ref.), 1954.
E – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – autocoids – *CAAAL-6902-D3 A-0498.

In court, a man pleaded guilty to the charge of being under the influence of "drink or a drug" while operating a motor vehicle. His doctor testified that the man had received antihistamine capsules for an allergic condition, and that he had not been warned of the possible effects of alcohol. On the night in question, the defendant had consumed a bottle of wine with 2 other persons, and one pink gin. He was fined and his license was endorsed, but the court acknowledged that there were special circumstances, and did not suspend his license.

45. Anonymous (Any Questions?)
ASPIRIN AND ALCOHOL.
Brit. Med. J. (London), 2: 1485-1486 (0 ref.), 1962.

E – general – DC (add., infra-add., unspec. incr.) – humans – CNS – metab. proc. – analg., antipyret.
 – *CAAAL-0 A-0499.

The question whether aspirin potentiates the action of alcohol when the two are taken simultaneously (in any amount) receives a negative reply. It is unlikely that aspirin could potentiate any of the several pharmacological actions of alcohol. There is no reason to suppose that ingestion of reasonable amounts of both would lead to a greater impairment of skill than would result from the alcohol alone. In cases of possible attempted suicide by combinations of large doses of each, it is doubtful that death would follow, since alcohol causes cerebral depression and aspirin a metabolic disturbance.

46. Anonymous (News and Notes. Medico-Legal: Coroners' Cases)
BARBITURATES AND ALCOHOL.
 Brit. Med. J. (London), 1: 1578 (0 ref.), 1960.
 E – general – DC (add., infra-add., unspec. incr.) – med.-leg. – drug-dep. humans – barbiturates –
 *CAAAL-0 A-0500.

A coroner's case is described in which a 47 yr-old patient died as a result of the "deadly combination" of barbiturates and alcohol while undergoing treatment for chronic alcoholism. He was allowed out of the hospital in the evenings, and on the fateful day had had his routine dose of 6 g sodium amytal upon his return to the dormitory. In his verdict, the coroner expressed his concern that steps should be taken to ensure that barbiturates are not given to patients who might have been drinking.

47. Anonymous (Any Questions?)
BARBITURATES AND ALCOHOL.
 Brit. Med. J. (London), 2: 877 (1 ref.), 1960.
 E – general – DC (add., infra-add., unspec. incr.) – humans – dose resp. – blood lev. – respir. –
 barbiturates – *CAAAL-9561-D1 A-0501.

In answer to the question, "To what extent does alcohol increase the respiratory depressive action of barbiturates?", it is stated that alcohol has been shown to potentiate the lethal effect of barbiturates in animals. In man, individual susceptibility to both drugs varies so much that definite dangerous combined dosages cannot be stated. Regarding a dosage of 3 g sodium amytal in combination with alcohol, the author states that, "It seems very improbable that 3 g of sodium amytal would increase the depression of the respiratory center produced by alcohol to the point of producing death unless very large doses of alcohol had been taken."

48. Anonymous (Queries and Minor Notes)
BEER AND PENTOBARBITAL (NEMBUTAL) AND A FRACTURED SKULL.
 J.A.M.A. (Chicago), 149: 1078 (0 ref.), 1952.
 E – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – barbiturates –
 *CAAAL-6167-D5 A-0502.

A question is asked concerning a man, aged 33, who was struck on acid-base, the head while working. At 2 p.m. he returned to work; at 4 p.m. he took 0.10 g of nembutal, and, at 5:30 p.m., he left to drive a truck. On the way he stopped and drank a bottle of beer. Afterwards he lost control of the truck and died in the resulting accident. Could the combination of alcohol and barbiturates make him so drunk as to render him reckless? The answer given is that the amount of alcohol in 1 bottle of beer could have caused at most a concentration of 0.02%—not enough to cause impairment. The barbiturate alone could have had sufficient sedative effect to have contributed to the accident, or the victim may have had an idiosyncrasy to the drug. The reply ignores altogether the possibility of a synergistic effect of the alcohol-barbiturate combination.

49. Anonymous (Any Questions?)
CHLORAL HYDRATE AND ALCOHOL.
Brit. Med. J. (London), 2: 923 (0 ref.), 1965.
E – general – DC (add., infra-add., unspec. incr.) – humans – CNS – respir. – sed., hypnot. –
*CAAAL-0 B-0240.

The question is asked, “Has chloral hydrate any synergistic action with alcohol?” The answer given is that chloral hydrate and ethanol have fairly similar actions on the central nervous system, and are likely to be at least additive in causing sedation, sleep, or respiratory failure. Whether either drug actually potentiates the other does not seem to have been tested experimentally, at least not in man.

50. Anonymous (Queries and Minor Notes)
CHLORAL HYDRATE AND WHISKY.
J.A.M.A. (Chicago), 113(25): 2259 (0 ref.), 1939.
E – general – DC (add., infra-add., unspec. incr.) – humans – CNS – liver, kidney – sed., hypnot.
– *CAAAL-0 A-0503.

In answer to a question regarding the safety of taking chloral hydrate with alcohol, the author states that the two should never be combined in a prescription, as they then form chloral alcoholate, which is a powerful and rapidly-acting central depressant. The patient in question, however, is taking single doses of chloral hydrate with his whiskey and seems to need this amount of depression to sleep because of his asthma.

51. Anonymous (Branch and Association News)
CHLORPROMAZINE AND ALCOHOL.
Pharmaceutical Journal (London), S₄ 121: 423 (0 ref.), 1955.
E – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – tranquilizers
– *CAAAL-0 A-0504.

A case is described of a man who pleaded guilty to driving under the influence of drink. On the evening of the offence, he had had three bottles of beer. As well, he was taking chlorpromazine, prescribed by his physician, who had not warned him of the possible dangers of drinking while under this medication. Because of these circumstances, his license was not suspended, although he was fined and his license was endorsed.

52. Anonymous (Queries and Minor Notes)
COMBINED ALCOHOL AND BARBITURATE INTOXICATION.
J.A.M.A. (Chicago), 148: 976 (0 ref.), 1952.
E – general – DC (add., infra-add., unspec. incr.) – humans – dose resp. – blood lev. –
*CAAAL-6051-E3 A-0505.

Question: A woman, 45 yr old, was found dead; the blood contained 0.27% alcohol by wt and 2.65 mg/100 cc barbituric acid derivative. Could this account for her death? Answer: Yes, although neither substance alone could have caused death in these concentrations. The mentioned concentration of a barbiturate would not be dangerous if resulting from a slow-acting one; if resulting from a fast-acting one, it could represent half a fatal dose. The mentioned alcohol concentration is a little over half of the presumably fatal dose (0.5%). But, since the effects of these two substances tend to be additive, death can be attributed to their combination.

53. Anonymous (Any Questions?)
DRINK AND DRUGS.
Brit. Med. J. (London), 1: 1555 (2 ref.), 1961.

E – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans
– CNS – autonomic agents – stimulants – tranquilizers – *CAAAL-0 A-0506.

A question is posed regarding the possible synergism of ephedrine and of drugs in general with alcohol, with regard to driving safety. The author states that it is dangerous for anyone to take any drug which has depressant effects on the central nervous system, e.g., barbiturates, tranquilizers, motion sickness preparations, etc., and to take any alcohol at all before driving. Stimulants, on the other hand, should tend to counteract the effects of alcohol. The author is then quick to state that he certainly is not advocating the use of stimulants in conjunction with alcohol; all drug-alcohol combinations should be avoided, especially in driving a motor vehicle.

54. Anonymous (Medical News)

DRUG MAY COUNTERACT DEPRESSION CAUSED BY ETHANOL INTOXICATION.

J.A.M.A. (Chicago), 189(12): 36-37 (0 ref.), 1964.

E – review – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – CNS – metab. proc. – nerv. syst. – respir. – skel., muscle, skin – amphetamines – hallucinogens – stimulants – *CAAAL-0 A-0507.

Experiments reported by Greenberg, R.E., Goldstein, L., and Pfeiffer, C.C., to the American Society for Pharmacology and Experimental Therapeutics (see also Greenberg, R.E., et al.: *Pharmacologist* (Detroit), 6(2): 170, 1964) are discussed. Acute ethanol intoxication was induced in rabbits, and the counteractive effects by CNS stimulants and tertiary amines were tested. Analysis of cortical EEG manifestations showed significant inhibition of ethanol-induced depression by dextroamphetamine, LSD, deanol, acetamidobenzoate, and desmethylecarnitine. Despite marked reduction in the relative degree of cortical EEG depression, all animals showed the same peripheral manifestations (marked nystagmus, loss of abdominal reflexes, decrease in muscle activity, respiratory depression) regardless of pretreatment.

55. Anonymous (Foreign Letters: England)

DRUGS AND DRIVING.

J.A.M.A. (Chicago), 156(6): 628-629 (0 ref.), 1954.

E – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – sed., hypnot. – *CAAAL-6903-N11 A-0508.

After several drinks of gin, a man took 20 capsules of oblivon (methylpentynol). Afterwards he had a car accident of which he remembered nothing. At the trial a physician testified that, in large doses, oblivon could accentuate the effects of alcohol. The defendant was convicted of driving under the influence of alcohol and drugs, was fined, and had his license withdrawn for a year. Soon after, the council of the Pharmaceutical Society recommended that the drug be sold on prescription only.

56. Anonymous (Any Questions?)

DRUGS, DRINK, AND DRIVING.

Brit. Med. J. (London), 2: 740 (0 ref.), 1964.

E – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – CNS – tranquilizers – *CAAAL-0 A-0509.

In answer to a query regarding the advisability of a patient, who is taking chlorpromazine (300 mg) and haloperidol (3 mg) daily, drinking alcohol or driving a car, the author replies in the negative. Any drug depressing the central nervous system will add to the depressing effects of ethanol. As well, if the patient should become involved in an accident, he might find himself liable to a charge of driving while under the influence of drink or drugs.

57. Anonymous (News and Notes. Medico-Legal)
DRUGS, DRINKING, AND DRIVING.
 Brit. Med. J. (London), 2: 776 (0 ref.), 1967.
 E – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – barbiturates
 – *CAAAL-0 B-0241.

The medico-legal implications of drinking while under medication and then driving a motor vehicle are discussed from the point of view of physician responsibility. A case is cited of a man who had been prescribed tablets heavily loaded with barbiturate, and, on the night in question, had taken 2 tablets and had drunk “a sensible modicum of whiskey”. Although he pleaded guilty to the charge of driving a car while unfit through drink or drugs, he was given an absolute discharge, since his doctor testified that he had not been warned of the danger of concomitant alcohol ingestion. It is noted that if a patient were badly injured in such a case as this, he would have a good cause for suing his physician for negligence. The duty of a physician in giving warning of possible hazards is thus a very high one.

58. Anonymous (Queries and Minor Notes)
EFFECT OF DRUGS ADDED TO ALCOHOLIC BEVERAGE.
 J.A.M.A. (Chicago), 133(16): 1253 (0 ref.), 1947.
 E – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – dose resp. – cardiovasc. – respir. – analg., antipyret. – anesthetics – barbiturates – *CAAAL-4622-D3
 A-0510.

In answer to a question regarding the effects of “doctoring” a whiskey and soda with small additions of a barbiturate, morphine, cocaine, or acetylsalicylic acid, the author replies that a specific answer is impossible, since effects depend on dosage of each of the drugs in question. The ordinary pharmacological effects of the various compounds would not be altered; the depressant effects of barbiturates would summate with the depressant effects of the alcohol, whereas cocaine, a stimulant, would probably prolong the excitatory phase of alcohol. The author stresses that there is no simple chemical test for the substances in question, and the material should best be placed for analysis in the hands of a competent toxicologist.

59. Anonymous (Foreign Letters: Berlin)
EFFECTS OF ALCOHOL, COFFEE AND TOBACCO ON MOTORISTS.
 J.A.M.A. (Chicago), 112: 1274 (0 ref.), 1939.
 E – exp. – general – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – cardiovasc. – stimulants – *CAAAL-0 A-0511.

A report is made of studies by Dr. Mueller, director of the Institute of Forensic Medicine at Heidelberg University. Human subjects drank substantial quantities of beer and then took very strong coffee (40 g of coffee beans of superior quality in 400 cc of boiling water, allowed to stand for 15 min). No change in the blood alcohol level was noted. The subjective and objective effects of alcohol were alleviated 15 to 20 min after the coffee; reaction time and the feeling of fatigue were diminished, and gait and speech improved. Nevertheless, recovery was of short duration and much diminished if the subject was accustomed to coffee; the recovery was followed by a state of such pronounced relaxation as to constitute a safety hazard in driving. If coffee drinking after alcohol consumption was accompanied by smoking, the sobering effect of coffee was in large part offset by the nicotine.

60. Anonymous (Queries and Minor Notes)
EFFECTS OF ALCOHOL ON WORKERS WITH CARBON DISULFIDE.
 J.A.M.A. (Chicago), 109(18): 1472-1473 (2 ref.), 1937.
 E – general – DC (sensit.) – humans – mammals – mot. perform. – psychol. perform. – cardiovasc. – miscellaneous – *CAAAL-0 A-1368.

A question is asked by a plant physician of a chemical company which manufactures tetramethylthiuram di- and monosulfides. Workers in the plant cannot drink any alcohol, or they suffer flushing of the face and hands, rapid pulse, and a terrible feeling of fullness in the face, eyes, and head. After 1-6 oz glass of beer, the blood pressure falls about 10 points, the pulse is slightly accelerated, and the skin becomes flushed in the face and wrists. In 15 min, the blood pressure falls another 10 points, the heartbeat is more rapid, and the patient complains of fullness in the head. As tests and observations of the questioner have not revealed any harmful effect, other than alcohol intolerance, of the chemicals on humans or mice, is it possible that they are a cure for alcoholism? The answer given is that alcohol may be regarded as a provocative agent, "demonstrating the actuality of clinical sulfide poisoning, minor in degree at the present time but possibly constituting much more of a threat to the well being of exposed workers than implied in the query." The skin-irritating properties of the chemicals, and the similarity of their action to that of carbon disulfide, are noted.

61. Anonymous

HEAVY DRINKING ACCELERATES DRUGS' BREAKDOWN IN LIVER.

J.A.M.A. (Chicago), 206(8): 1709 (0 ref.),

1968.

E – review – exp. cont. – exp. comp. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – metab. proc. – anticonvulsants – coagulants – hormones, hormone antag. – *CAAAL-0

B-0242.

Experiments reported by R.M.H. Kater to the American Association for the Study of Liver Diseases are discussed. Tests were carried out to determine the increase in metabolism of several drugs in clinical use. Heavy drinkers and control subjects each received, in three separate tests: 1 g tolbutamide iv, 3-100 g doses diphenylhydantoin daily for 3 days, or 40 mg warfarin in one single dose. Heavy drinkers were considered persons who consumed a minimum of 200 g ethanol daily. The drinkers underwent a drying-out period before the tests started. The results were: for tolbutamide, a half-life of 165 min for drinkers versus 351 min for control; for diphenylhydantoin, drinkers 16.3 hr and non-drinkers 23.5 hr; and for warfarin, drinkers 26.5 hr and non-drinkers 41.0 hr.

62. Anonymous (Queries and Minor Notes)

METHYL ALCOHOL INTOXICATION.

J.A.M.A. (Chicago), 138(3): 253 (4 ref.),

1948.

E – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – med.-leg. – mot. vehic. – humans – absorp., distrib., stor. – metab. proc. – alcohols – analg., antipyret. – autonomic agents – *CAAAL-5045-E3

A-0512.

A question is asked concerning a man, about 50 yr old, who had taken some whiskey the previous evening and also on the following morning, before having several teeth extracted. Soon afterwards he took some acetylsalicylic acid tablets and drove a car in which the odour of the methyl alcohol antifreeze was strong. He was arrested in a very drunken state but recovered quickly. It is asked, would the fact that he had taken whiskey make the effect of the fumes greater? Would the effects of the whiskey, antifreeze, and acetylsalicylic acid cause greater impairment than from alcohol alone? The reply given states that it is probable that the man was still under the influence of the whiskey consumed in the morning, and, since ethanol prevents methanol oxidation, it is unlikely that the antifreeze fumes were an important factor. The acetylsalicylic acid would not have affected the condition unless taken with codeine, and, if so, the effect would still have been negligible. The extraction of the teeth may have had some effect, especially if he had been given procaine hydrochloride with epinephrine.

63. Anonymous

OPIUM AND ALCOHOL: THEIR COMPARATIVE EFFECTS ON THE SYSTEM, DESCRIBED BY ONE WHO EXPERIENCED THEM IN HIS OWN CASE.

New York Medical Times (New York), 3(2): 37-46 (0 ref.), 1853.
 E – SEC – general – case hist. – conj. addict. – DC (add., infra-add., unspec. incr.) – drug-dep. humans
 – mot. perform. – psychol. perform. – CNS – hallucinogens – *CAAAL-0 A-1243.

In this classic, lucid description of opium and alcohol addiction, an addict recounts the effects that the chronic ingestion of the substances have had on his body and mind. It is explained that, whereas alcohol tends to dull the intellect and bring the “lower” faculties into undue prominence, opium invigorates the intellectual and affectional faculties, while depressing the vegetative appetites, such as the desires for bodily exercise and sexual experiences. After a 3 1/2 yr history of alcoholism, the writer switched to laudanum, taking approximately 20 drops, 2 or 3 times a day. After a considerable time, the opium pill was substituted for the laudanum, and 1/2 oz/week was consumed for a 3 yr period. Toward the end of this period, he deteriorated into a physical state which opium would not stimulate, and felt compelled to employ alcohol. According to the author, the alcohol, acting on his opium-drugged nerves, had a strong tendency to produce a maniacal intoxication. Except for brief periods, he continued his use of opium (1/2 oz/week) or morphine (1/4 oz/week) until the time of this narration.

64. Anonymous (Any Questions?)
 ORAL ANTIBIOTICS, ALCOHOL, AND ASPIRIN.
 Brit. Med. J. (London), 1: 864-865 (1 ref.), 1963.
 E – general – DC (unchanged) – humans – dose resp. – absorp., distrib., stor. – G.I. tract –
 anti-infectants – *CAAAL-0 A-0513.

In answer to a query regarding the efficacy of oral antibiotics in the presence of alcohol or aspirin, the author replies that it is unlikely that the presence of moderate amounts of alcohol or aspirin in the stomach would have any appreciable effect upon the efficacy of either phenoxymethyl-penicillin, or tetracycline. If large amounts of alcohol are taken, there may be a cessation of gastric activity and, hence, a delay of absorption of any drug taken orally.

65. Anonymous (Any Questions?)
 PENICILLIN AND ALCOHOL.
 Brit. Med. J. (London), 1: 1462 (0 ref.), 1963.
 E – general – DC (unchanged) – humans – blood lev. – anti-infectants – *CAAAL-0 A-0514.

In answer to a query regarding the possibility of inactivation of penicillin by a high blood alcohol level, it is stated that there is no reason whatever for supposing that the relatively low alcohol concentrations occurring in the blood, even in alcoholic coma, would have any deleterious action.

66. Anonymous (Any Questions?)
 PHENOBARBITONE AND ALCOHOL.
 Brit. Med. J. (London), 2: 35 (8 ref.), 1966.
 E – general – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – humans – barbiturates
 – *CAAAL-0 B-0243.

In answer to the question, “Are the clinical effects of alcohol and barbiturates synergistic or additive?”, the author points out that, while some tests on animals indicate a potentiating effect, other researchers claim that, if allowance is made for the different times of action of the depressant drugs and the different methods of administration, the balance of evidence is in favour of a simple additive effect. In humans the data suggests a potentiating effect, although this is a complex matter of some dispute.

67. Anonymous
 THE POISONOUS COÖPERATION OF ALCOHOL AND TOBACCO.
 Lancet (London), 162: 327-328 (0 ref.), 1902.
 E – general – DC (add., infra-add., unspec. incr.) – humans – absorp., distrib., stor. – *CAAAL-0
 A-0515.

The author describes why the drinking of alcohol after heavy smoking is particularly detrimental to health. He states that the chief poisonous constituents in tobacco are pyridine bases, and they are very soluble in alcohol but not water. An alcoholic drink, therefore, washes the pyridine into the stomach where it is absorbed, giving rise to definite toxic symptoms due to combined effects of both.

68. Anonymous (Panel on Interpretation and Medical Aspects)
 QUESTIONS AND ANSWERS. [ANSWERS TO A QUESTION CONCERNING
 TRANQUILIZERS]
 In: Myren, Richard A., ed. *Proceedings of the Symposium on Alcohol and Road Traffic*. Held at
 Indiana University, December 12-14, 1958. Bloomington, Indiana: Indiana University Press, pp.
 224-227 (0 ref.), 1959.
 E – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – other
 drug lev. – psychol. perform. – CNS – amphetamines – tranquilizers – *CAAAL-0 A-0516.

In answer to a query regarding the effect of tranquilizers on drivers and of the availability of chemical tests for their detection, the consensus of opinion seems to be that they can, in general, impair some faculties necessary for safe driving. This effect would summate with the deleterious effects of alcohol on the driving situation. These drugs do not interfere with the measurement of blood alcohol levels. An opinion is also expressed that drugs such as benzedrine, which might be considered effective in offsetting the effects of alcohol, have in fact an effect which is an additional impairment.

69. Anonymous (Questions and Answers)
 REACTION TO ALCOHOL AND CHLORAL HYDRATE.
 J.A.M.A. (Chicago), 167(2): 273 (0 ref.), 1958.
 E – general – DC (sensit.) – humans – cardiovasc. – skel., muscle, skin – sed., hypnot. –
 *CAAAL-8458-E3 A-0517.

A query is made regarding the idiosyncratic reaction of a patient who, when drinking any alcohol up to three days after his last dose of chloral hydrate (1 or 2 g), becomes uncomfortably violently red in the face. The author replies that this response, similar to a disulfiram reaction, is not characteristic of either drug, and may be the result of some undiagnosed liver or kidney disturbance.

70. Anonymous (Queries and Minor Notes)
 REACTION TO SMALL AMOUNT OF ALCOHOL.
 J.A.M.A. (Chicago), 143: 1040 (0 ref.), 1950.
 E – general – case hist. – DC (antidotal) – mot. perform. – cardiovasc. – G.I. tract – glands – autotoxics
 – autonomic agents – *CAAAL-5489-C3 A-1251.

A question is asked regarding a man who is neither an alcoholic nor inclined to excessive drinking, and yet, after small doses of alcohol, develops symptoms of a much higher degree of intoxication. In a test, he was given 385 cc of alcohol. 15 min later the symptoms appeared, and he was given 50 mg of diphenhydramine. No change occurred in his condition. 30 min later he received 0.5 cc of epinephrine, after which the symptoms subsided. The reply given offers 3 possible explanations: the man may have had a low pharmacological tolerance towards alcohol; there could have been an alcohol-stimulated release of histamine, inducing allergy-like effects; or an already existing allergy may have been precipitated by the vasodilating effect of alcohol. It is suggested that an antihistaminic

drug be given in a fairly large dose 1 hr before the ingestion of alcohol. If this therapy proves to be ineffective, administration of a vasoconstrictor, such as ephedrine (25 mg), 1 hr before ingestion of alcohol could be tried.

71. Anonymous
REMEDY FOR DRUNKENNESS OUTLAWED UNDER PURE FOOD AND DRUG LAWS.

Quart. J. Stud. Alcohol (New Haven), 2: 607 (0 ref.), 1941.
E – general – DC (unchanged) – drug-dep. humans – *CAAAL-0 A-0518.

The results of a court action against a remedy for drunkenness are presented. The remedy was condemned by a United States District Court under the Federal Food, Drug, and Cosmetic Act. The remedy contained 3.2 grains of potassium antimony tartrate (tartar emetic), and five doctors testified this was enough to be considered a poison—in no way was it a cure for drunkenness. For 60 years the company producing the substance sold about 50,000 powders a year.

72. Anonymous (Any Questions?)
SOBERING UP WITH ASPIRIN.

Brit. Med. J. (London), 1: 1399 (0 ref.), 1955.
E – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – other drug lev. – amphetamines – analg., antipyret. – sed., hypnot. – *CAAAL-7278-N11 A-0519.

In answer to a query, it is stated that acetylsalicylic acid (aspirin) does not alter the symptoms of drunkenness and does not affect the concentration of alcohol in the blood. Oblivon (methylpentynol) would tend to increase the apparent degree of intoxication. Amphetamine, taken after a person has stopped to drink, has a distinct sobering effect; taken the morning after, it prevents or abates the usual symptoms of hangover.

73. Anonymous
A STRYCHNIA-EATER.

Lancet (London), 1: 669 (0 ref.), 1875.
E – general – DC (decrease) – humans – skel., muscle, skin – stimulants – *CAAAL-0 A-0520.

The following account is given of a strychnine user. The individual in question was a male, subject to fits of intemperance. He would go on sprees which lasted from one to three weeks, during which time he kept completely saturated with whiskey. When he wanted to sober up, he would take from 10 to 20 grains of strychnine, and, no matter how much he drank, within 3 hr all trace of debauch left him. "The closest observer could not discover the slightest indication of recent dissipation. Instead of a hectic flush, a dull, heavy look, his eyes are clear and bright, and his skin presents its natural appearance."

74. Anonymous (Notes)
STRYCHNINE NOT A SAFE MEDICINE.

Journal of Inebriety (Hartford), 34(1): 48-49 (0 ref.), 1912.
E – general – DC (unspec.) – humans – CNS – skel., muscle, skin – stimulants – *CAAAL-0 A-0521.

A warning is given of the dangers of using strychnine for the treatment of inebriety. Reference is made to studies which show that a certain number of people are hypersensitive to its effects (1 or 2 doses, 1-60 grains) and experience a spasmodic action and muscular tension that is very dangerous. Where

the doses are smaller and its use extends over several weeks, there appears an inexplicable sort of dementia. The author adds that, in toxemias and congestions from alcohol and inebriety, strychnine should be used with great caution and careful examination of its effects, otherwise much danger will follow.

75. Anonymous (Today's Drugs)

TWO HYPNOTICS.

Brit. Med. J. (London), 2: 1608 (0 ref.),

1962.

E – SEC – DC (add., infra-add., unspec. incr.) – psychot. humans – sed., hypnot. – *CAAAL-0 A-0522.

A study by T.A. Ban and K. McGinnis at Verdun Protestant Hospital, Quebec, is reported. The time needed to fall asleep, and the amount of sleep, were measured in a double-blind, six-day experiment on 20 chronic psychiatric patients who were given either glutethimide or ethchlorvynol. The results of the experiment are discussed, and the dangers of a combined intake of ethchlorvynol and alcohol are stressed.

76. Arbuzov, S. Ia.

ANTAGONIZM FENAMINA, KORAZOLA I IKH SMESI PO OTNOSHENIIU K METILOVOMU I ETILOVOMU SPIRTAM I ETILENGLIKOLIU (ANTIFRIZU).

[Antagonism between phenamine, corazol, and their mixture to methyl and ethyl alcohols and to ethylene glycol (antifreeze)].

Fiziol. Zh. S.S.S.R. Sechenov. (Moscow), 38(3): 337-343 (24 ref.),

1952.

R – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – CNS – alcohols – amphetamines – stimulants – *CAAAL-6376-D2 A-0523.

Lethal doses (10 ml/kg) of ethyl alcohol, methyl alcohol, or ethylene glycol were administered to rabbits by stomach tube. Antagonists of narcotics—phenamine (0.0005-0.001 g/kg), corazol (0.005-0.01 g/kg), or a mixture of both—given iv during the first 24 hr of intoxication prevented death. The restoration of body temperature, slowed by the administration of intoxicants, proceeded parallel to the arousal of subjects from narcosis under the influence of analeptics.

77. Archer, J. D.

AN EXPERIMENTAL STUDY OF THE LETHAL SYNERGISM BETWEEN SECOBARBITAL AND ALCOHOL.

Texas Rep. Biol. Med. (Galveston), 14: 1-5 (13 ref.),

1956.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – barbiturates – *CAAAL-7584-D2 A-0524.

The LD₅₀'s of secobarbital and ethanol were determined in mice; the sol were administered ip, ethanol as a 20% sol. The LD₅₀ for secobarbital was 126.5 ± 1.9 mg/kg; for ethanol it was 9.3 ± 0.4 ml/kg. An additive synergistic action of the two drugs was confirmed. A potentiating synergism may occasionally occur in a susceptible individual.

78. Arima, Y.

ZOKI-SHŌGAIJI NI OKERU KETSUEKI-SHUSEIRYŌ NO SHŌCHŌ NI KANSURU JIKKENTEKI KENKYŪ. [Investigations of the blood alcohol level during experimental functional impairment of the organs].

Fukuoka Acta Medica (Fukuoka Igaku Zasshi) (Fukuoka), 34(3): 249-286 (88 ref.),

1941.

J – exp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – cardiovasc. – liver, kidney – anti-infectants – miscellaneous – *CAAAL-O A-1307.

To study the effect on alcohol metabolism of drug-induced damage to internal organs, male rabbits (2 kg) received a single dose of 8 cc/kg 20% ethanol po. 1-2 weeks later, a second ethanol dose was administered under the following conditions: liver damage induced by carbon tetrachloride sc, kidney damage induced by potassium chromate or cantharidin sc, heart damage produced by paraffin infusion into the pericardium, or lung damage produced by infusion of lamp black sol into the tracheae. Blood alcohol determinations were made by the Widmark micromethod, and the values compared with those found after the previous alcohol administration. It was found that carbon tetrachloride retarded the decline of the blood alcohol level (BAL), and produced higher alcohol concentrations. Potassium chromate and cantharidin failed to affect the BAL. Paraffin and lamp black sol resulted in an accelerated decrease of and lower BAL. The author concludes that determinations of the BAL should always take into account possible damage of internal organs.

79. Ariyama, H., and Takahasi, K.
ÜBER DEN RELATIVEN NÄHRWERT DER KOHLENHYDRATE UND VERWANDTEN SUBSTANZEN. [The relative nutritional value of carbohydrates and related substances].
Biochemische Zeitschrift (Berlin), 216: 269-277 (1 ref.), 1929.
G – SEC – exp. comp. – congen. stud. – mammals – chronic admin. – in vivo – nutritive agents – *CAAAL-1383-A2 A-1369.

The relative nutritional values and assimilability of carbohydrates and other related substances (total of 28 substances) were determined in rats. In 1 experiment, young male rats (40-45 g) received 4.2 g/day of a basic diet (27% meat powder, 70% butter, and 3% McCollum's salt mixture, plus an addition of 0.2 g dry yeast/day) until growth stopped (average weight of 80 g after 41 days), then 1.25 g/day of test feed were given in addition to the basic diet for 40 more days. Various substances, including ethyl alcohol and cognac, were added to the test feed. The results were tabulated and graphically compared. Ethanol caused the greatest weight increase of 49 g over 40 days (average gain of 1.23 g/day), compared to an increase of 19.5 g in rats fed cognac (average gain of 0.49 g/day). The rats consumed the alcohol feed without signs of intoxication or narcosis, and remained lively and healthy. The animals had a high tolerance towards alcohol, and dissection revealed no pathological organ changes.

80. Ariyoshi, T., Takabatake, E., and Remmer, H.
DRUG METABOLISM IN ETHANOL INDUCED FATTY LIVER.
Life Sci. (Oxford), 9(2): 361-369 (20 ref.), 1970.
E – exp. cont. – cross-tol. – mammals – acute admin. – chronic admin. – in vivo – liver, kidney – metab. proc. – sed., hypnot. – *CAAAL-0 B-0909.

The effects of ethanol on hepatic lipids, drug-metabolizing enzyme activities, and cytochrome P₄₅₀ and b₅ were investigated in male and female Wistar rats. The inductive effect of ethanol was also compared with that of pentobarbital. The rats were maintained on normal and on choline-deficient diets for 20 days. Ethanol was administered acutely (5.0 g/25 ml/kg body wt by stomach tube 16 hr before sacrifice) or chronically (20% sol as drinking water). Livers from rats in each group were pooled and homogenized for analysis. The single dose of ethanol produced a 50% decrease in coenzyme A and an 80% increase of triglycerides in liver; aniline hydroxylase activity was increased, but that of aminopyrine demethylase was not. Results with P₄₅₀ and b₅ showed no change. With respect to chronic ethanol administration, the triglyceride level in the choline-deficient-ethanol group was about 5 times higher than in the normal diet-ethanol group. Although choline deficiency itself did not change the activities of the drug-metabolizing enzymes, these activities were markedly increased by ethanol in both groups. The effect of ethanol was more remarkable in choline-deficient than in normal rats. P₄₅₀ and b₅ concentrations were enhanced. It is suggested that alcohol causes qualitative and quantitative changes in the smooth endoplasmic reticulum membrane, and consequently induces change of drug-metabolizing enzyme activities.

81. Ariyoshi, T., and Takabatake, E.
 DRUG METABOLISM IN ETHIONINE INDUCED FATTY LIVER.
 Life Sciences (New York), 9(2): 371-377 (21 ref.), 1970.
 E – exp. cont. – DC (decrease) – DC (supra-add. incr.) – DC (unchanged) – mammals – acute admin.
 – in vivo – liver, kidney – metab. proc. – *CAAAL-0 B-0910.

Female rats were used to compare the effect on the drug metabolizing system in fatty livers induced by ethionine, with that induced by ethanol. Ethionine was injected ip in 3 doses at 2 hr intervals to a total of 750 mg/kg. Ethanol was given orally, 5.0 g/25 ml/kg, 16 hr before sacrifice. Phenobarbital was injected ip, 80 mg/2 ml/kg, 24 hr before sacrifice. In the case of combined treatments (ethionine and phenobarbital, or ethionine and ethanol) ethionine was given 2 hr before phenobarbital or ethanol. Rats were killed 6, 12, 24, 48, 72, and 120 hr after last ethionine injection. Livers were pooled in groups and homogenized for analysis. Ethionine caused a reduction in cytochrome P_{450} and b_5 and in the spectral changes with aniline, compared to controls. Microsomal drug-metabolizing activities did not parallel the P_{450} levels. Pretreatment with ethionine completely abolished the increase by phenobarbital or ethanol of the contents of cytochromes and the spectral change. However, the stimulation by ethanol of aniline hydroxylase was not affected by ethionine pretreatment. The increase in triglycerides (TGL) induced by ethanol was synergistically enhanced by ethionine. Kinetic analysis showed that ethionine and ethanol each induced increased liver TGL and aniline hydroxylase, but that they affected cytochrome P_{450} and b_5 differently.

82. Armour, P. S.
 A REGIMEN IN ACUTE ALCOHOLISM.
 Arizona Med. (Scottsdale), 13(5): 169-172 (9 ref.), 1956.
 E – exp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – drug-dep. humans
 – chronic admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – cardiovasc. – G.I. tract
 – respir. – musculoskel. agents – tranquilizers – *CAAAL-7565-N47 A-1253.

49 out of 100 acute alcoholics were treated with reserpine (5-60 mg/day im) and mephenesin carbamate (1.5 oz po/day) concomitantly, 14 with mephenesin carbamate alone, and 37 with reserpine alone. Mephenesin carbamate, characterized by profound muscle relaxant properties with an associated tranquilizing or sedative effect, was administered in an elixir form containing dimenhydrinate to minimize gastrointestinal side-effects. Both drugs were contraindicated if the blood pressure was below 120/70. 29 of the 49 patients (59%) receiving reserpine-mephenesin carbamate therapy had good to excellent results, as shown by marked quieting or relaxing effects, and 8 patients had fair results in which persistent side-effects required additional medication, such as barbiturate sedation. 6 of 14 (43%) receiving reserpine alone showed marked improvement, as did 22 of 37 (59%) receiving mephenesin carbamate alone. Side effects were minimal. There was no evidence that either reserpine or mephenesin carbamate potentiate alcohol effects. However, many patients with very high blood alcohol levels, as well as indeterminate amounts of barbiturate when admitted, experienced a physiological type of sleep for as long as 12-20 hr after a parenteral dose of 5-10 mg reserpine.

83. Arnold, W.
 UNBEMERKTE BEIBRINGUNG VON SCHLAFMITTELN IN ALKOHOLISCHEN
 GETRÄNKEN: NEUE GANGSTERMETHODEN AUF ST. PAULI. [Undetected
 administration of hypnotics in alcoholic beverages: new gangster methods at St. Pauli].
 Kriminalistik (Stuttgart), 20: 363 (8 ref.), 1966.
 G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – other drug lev. – mot.
 perform. – sed., hypnot. – *CAAAL-0 B-0911.

The possibility of undetected administration, for criminal purposes, of drugs in alcoholic beverages is discussed, with special reference to noludar. Noludar, a commonly-available hypnotic, can be added without detection to aromatic alcoholic beverages, to make a victim easily susceptible to robbery or

fraud. A case is reported of a workman who was befriended by an unknown man. Already intoxicated by beer and cognac, the victim was given a drink containing a drug; he became almost unconscious, and awoke the next morning to find that he had been robbed. A urine analysis revealed the presence of a nolidar metabolite. The author points out that administration of even 1 ml (4 ml = 1 g) of nolidar leads to increasing exhaustion, dizziness, and finally sleep, rendering the victim virtually helpless and unaware of the situation. The dangers of the combination of hypnotics with alcohol are stressed—hypnotic doses just slightly higher than therapeutic can be fatal, even when given in combination with only small amounts of alcohol.

84. Aron, T.
EXPERIMENTELLE STUDIEN ÜBER SCHLANGENGIFT. [Experimental studies on snake poison].
Zeitschrift für Klinische Medizin (Berlin), 6(4): 332-360 (2 ref.), 1883.
 G – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – cardiovasc. – *CAAAL-0 A-0525.

The effect of alcohol as an antidote to snake bite was examined in controlled experiments on rabbits. Two rabbits (A and B) were employed for the test, one serving as control. The rabbit designated as A received 3 cc absolute alcohol with 7 cc water sc, followed by another injection (1 cc alcohol with 3 cc water) 10 min later. 3 min later both rabbits were injected with 1 cc 1% cobra poison sol sc. 22 min later, A received a further injection of cobra poison (0.2 cc). Both animals died, the control expiring 6 min earlier than A. The experimenter speculates that the longer longevity of A may be attributed to the action of ethanol (the alcoholized animal showed longer heart function than that of the control). In two subsequent tests, alcohol proved a poor antagonist, but prolonged life by 30 min at 1/2 the dose of cobra poison.

85. Arullani, C.
IL COMPORTAMENTO DELLA CURVA ALCOLEMICA IN SEGUITO A SOMMINISTRAZIONE DI ASPIRINA O DI CAFFEINA. [The behaviour of the blood alcohol curve in subjects treated with aspirin and caffeine].
Boll. Soc. Ital. Biol. Sper. (Naples), 17: 46-48 (0 ref.), 1942.
 I – exp. cont. – exp. comp. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – metab. proc. – analg., antipyret. – stimulants – *CAAAL-0 A-0526.

Tabulated data are presented of a series of controlled experiments with healthy young human subjects given either 1.5 g aspirin po 30 min before ingestion of 34 cc alcohol, or 25 cg caffeine administered hypodermically 10 min prior to alcohol (0.5 g/kg in 20% sol). For control, 34 cc alcohol (0.5 g/kg in 20% sol) was given alone. Widmark's micromethod modified by Baglioni was used to determine the blood alcohol level. The blood alcohol values obtained 30 min after alcohol ingestion were: 0.55 mg/ml for alcohol alone, 0.57 mg/ml for aspirin plus alcohol, and 0.76 mg/ml for caffeine plus alcohol. It is concluded that caffeine probably exercises a stimulating effect on alcohol metabolism, whereas no hypothesis is given for the action of aspirin as an inhibitor of acute alcohol intoxication (40 min after alcohol ingestion, the values for aspirin were 0.54 mg/ml, and 0.68 mg/ml for alcohol alone).

86. Arvola, A., Sammalisto, L., and Wallgren, H.
A TEST FOR LEVEL OF ALCOHOL INTOXICATION IN THE RAT.
Quart. J. Stud. Alcohol (New Haven), 19: 563-572 (6 ref.), 1958.
 E – SEC – exp. – mammals – mot. perform. – species or sex diff. – *CAAAL-8287-J2 A-0527.

In alcohol intoxication tests, effects on the sense of equilibrium and motor coordination were studied in rats. The sliding angle on a tilted plane with a rough surface was found to be the most reliable

testing apparatus. Five intoxication levels could be distinguished with doses ranging from 0 to 7.2 mg/g of orally administered alcohol. A technique suitable for determining whether or not drugs are synergistic with, or antagonistic to alcohol is described.

87. Aschan, G., Bergstedt, M., and Goldberg, L.
 THE EFFECT OF SOME ANTIHISTAMINIC DRUGS ON POSITIONAL ALCOHOL
 NYSTAGMUS.
 Acta Otolaryng. (Stockholm),, Suppl. 140: 79-90 (11 ref.), 1958.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged)
 – humans – acute admin. – in vivo – blood lev. – CNS – nerv. syst. – senses – amphetamines –
 autocoids – gastrointest. agents – sed., hypnot. – *CAAAL-8797-D1 A-0528.

87 experiments on 36 healthy humans showed that some antihistaminic drugs—tripelennamine (50 mg), chlorcyclizine (50 mg), meclizine (50 mg), promethazine (25 mg), amphetamine (10 mg), and promethazine (15 mg) plus amphetamine (10 mg)—can diminish or even eliminate phase II of positional alcohol nystagmus, and can diminish or eliminate certain subjective symptoms, such as nausea, vertigo, and vomiting, at least for some time. Some drugs accomplish this without sedation, and others exert an additional sedative effect.

88. Aschkenasy-Lelu, P.
 ACTION D'UNE INGESTION MODÉRÉE ET PROLONGÉE D'ALCOOL OU DE VIN
 SUR LA CROISSANCE ET LE POIDS RELATIF DES ORGANES DU RAT MÂLE. [Effect
 of moderate and prolonged ingestion of alcohol or wine on the growth and relative weight of
 organs of the male rat].
 J. Physiol. (Paris), 47: 78-80 (0 ref.), 1955.
 F – exp. cont. – congen. stud. – mammals – chronic admin. – in vivo – cardiovasc. – glands – liver,
 kidney – skel., muscle, skin – *CAAAL-7891-D2 A-1370.

34 male Wister rats were divided into 3 groups. When the animals were 30 days old, they were placed on an identical solid-food diet. All animals received 15 cc liquid/rat/day, the first group (control group) being given water, the second group (alcohol group) 5% alcohol diluted with water, and the third group (wine group) 10% red wine diluted with water. After 250 days of this regimen, the animals were extensively examined. Body growth and weight were normal in all groups. Relative weights of body, heart, liver, spleen, thymus, adrenals, preputial glands, seminal vesicles, salivary glands, and gastrocnemius muscle were virtually identical. The thyroid gland was slightly enlarged, and the testicles and kidneys slightly smaller, in the alcohol group, but the differences were not statistically significant. The prostate gland was significantly hypertrophied in the alcohol group; the wine group, on the other hand, was identical to the controls. A histological study of the prostates of all 3 groups failed to reveal any specific anomaly which might account for the hypertrophy, although an enlargement of connective interacinar tissue was observed in the alcohol group. This fibrous reaction was also noted in the majority of the wine group, despite the absence of the prostate hypertrophy.

89. Asser, E.
 ÜBER ÄNDERUNG DER METHYLALKOHOLOXYDATION DURCH ANDERE ALKOHOLE.
 [On the change of methyl alcohol oxidation caused by other alcohols].
 Dissertation, Medical Faculty of the University of Breslau, Germany, 13 pp. (9 ref.), 1914.
 G – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – other drug lev.
 – alcohols – *CAAAL-0 A-0529.

The influence of alcohols, including ethanol (25 cc), on the oxidation of methanol was determined in a series of controlled experiments. Ethanol (25 cc) was fed to a dog (8 1/2 kg) in combination with methanol (25 cc) by means of a probang in one test, and, in another, 20 cc methanol was combined

with 15 cc ethanol, with equal results. The precipitation of formic acid in the urine was 1.9360 g, compared with 3.1-4.6 g for pure methanol only; i.e., the quantity of formic acid receded with the ethanol intake. The combined intake showed no increase in intoxication symptoms, compared with methanol alone—with respect to outward behaviour of the animals.

90. Asser, E.
UEBER AENDERUNG DER METHYLALKOHOLOXYDATION DURCH ANDERE ALKOHOLE. [The effects of other alcohols on the oxidation of methanol].
Zeitschrift für Experimentelle Pathologie und Therapie (Berlin), 15: 322-334 (9 ref.), 1914.
G – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – chronic admin. – in vivo
– other drug lev. – metab. proc. – alcohols – miscellaneous – *CAAAL-O A-1329.

Dogs received various doses of methanol po, alone or in combination with varying doses of amyl alcohol, ethanol, or acetone. The oxidation of methanol was determined by measurement of formic acid concentration in the urine. All 3 test substances lowered formic acid concentration by about 50%. In another test, it was found that neither glucose nor glycerin significantly lowered the formic acid concentration after methanol administration. When sodium formate was administered to a group of animals, an average of 18% formic acid was found; when sodium formate was given with amyl alcohol, ethanol, or acetone, the percentage dropped to 9.5%, 4.5%, and 13.2%, respectively. In 1 test, alcohol breath levels were measured for 23 hr, and it was found that 7.98% of the alcohol was exhaled; this was unaltered by simultaneous sodium formate administration. To test chronic effects, the animals were given, over a 9-day period, 270 cc ethanol plus 48 cc amyl alcohol. When methanol was administered on the tenth day, the formic acid concentration was found to be 1/3 the control value. A similar test using sodium formate instead of methanol showed a formic acid concentration of 1/4 the control value. In conclusion, it is suggested that ethanol increases the oxidation of formic acid.

91. Astley, C. E.
GASTRITIS, ASPIRIN, AND ALCOHOL.
Brit. Med. J. (London), 4: 484 (1 ref.), 1967.
E – general – stat. surv. – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – G.I. tract – analg., antipyret. – *CAAAL-0 B-0244.

The author points out the considerable risk of gastro-duodenal hemorrhage when alcohol and aspirin are taken together. Of 31 men admitted with haematemesis and melena to the North Ormesby Hospital, Middlesbrough, England, 23 had taken aspirin and 16 alcohol (10 had taken both) within 24 hr of onset. Of 75 men with gastro-duodenal bleeding, 48 were heavy drinkers, whose consumption, nearly always of bitter beer, varied from 1 to 7 gallons/week. Of the 48, 32 had ulcers. The author reasserts his contention that a positive correlation exists between beer drinking (particularly bitter beer), ingestion of aspirin, and duodenal ulcers.

92. Aston, R., and Cullumbine, H.
STUDIES ON THE NATURE OF THE JOINT ACTION OF ETHANOL AND BARBITURATES.
Toxic. Appl. Pharmacol. (New York), 1: 65-72 (13 ref.), 1959.
E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – species or sex diff. – absorp., distrib., stor. – respir. – barbiturates – *CAAAL-9172-D2 A-0530.

The 24-hr LD₅₀'s were established in mice for 30% ethanol ip (6092.6 mg/kg), 50% ethanol ip (3744.5 mg/kg), 30% ethanol iv (2278.3 mg/kg), secobarbitone ip (115.6 mg/kg), and phenobarbitone ip (272.2 mg/kg). The 30% ethanol iv dose was not significantly affected by prior administration of 1/2

LD₅₀ phenobarbitone ip (2470.6 mg/kg) or 1/2 LD₅₀ secobarbitone ip (2086.4 mg/kg). The 30% ethanol ip dose was slightly increased by 1/2 LD₅₀ phenobarbitone ip (6464.0 mg/kg) and slightly decreased by 1/2 LD₅₀ secobarbitone ip (5560.4 mg/kg). It is concluded that the above combinations manifest a "negatively correlated independent additive joint action." The results of iv infusion of ethanol and pentobarbitone, individually and in combination in the rabbit, give added confirmation that ethanol interacts with barbiturates in an independent additive manner.

93. Aston, R., and Cullumbine, H.

THE EFFECTS OF COMBINATIONS OF ATARAXICS WITH HYPNOTICS, LSD AND IPRONIAZID IN THE MOUSE.

Arch. Int. Pharmacodyn. (Gand), 126(1-2): 219-227 (19 ref.), 1960.
 E – exp. cont. – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
 – dose resp. – other drug lev. – CNS – nerv. syst. – anesthetics – barbiturates – hallucinogens –
 tranquilizers – *CAAAL-0 A-1371.

The effects of ip administration of 5-hydroxytryptamine (5-HT) and 5 chemically-unrelated ataraxics (reserpine, chlorpromazine HCl, azacyclonol HCl, meprobamate, and SKF-183A) on hexobarbitone sodium and ethanol anesthesia, the effect of LSD on the duration of anesthesia produced by these drug combinations, and the effect of iproniazid on the responses to the ataraxics, were studied in mice. All drugs prolonged hexobarbitone hypnosis. Sleeping time due to 5 mg/kg ethanol as a 50% sol was significantly prolonged by 20 mg/kg 5-HT, 0.5 mg/kg reserpine, 5 mg/kg chlorpromazine, 80 and 100 mg/kg SKF-183A, and 80 and 100 mg/kg meprobamate, but not by azacyclonol in doses up to 100 mg/kg; however, with 150 mg/kg azacyclonol, immediate respiratory arrest occurred: LSD (0.001 mg/kg) blocked potentiation of ethanol hypnosis only in the case of 5-HT and reserpine. Pretreatment with iproniazid intensified the action of chlorpromazine, and caused slight excitation by 5-HT. Differences in sleeping times following hexobarbitone and ethanol were noted between 2 strains of mice.

94. Aston, R., and Stolman, S.

INFLUENCE OF ROUTE AND CONCENTRATION OF ETHANOL UPON CENTRAL DEPRESSANT EFFECT IN THE MOUSE.

Proc. Soc. Exp. Biol. Med. (New York), 123: 496-498 (7 ref.), 1966.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – absorp., distrib., stor. – CNS – barbiturates – *CAAAL-0
 B-0245.

The central depressant effect of ethanol was assessed in male mice in terms of the extent of prolongation of hexobarbital sleeping time produced. The dosage of alcohol was varied in terms of both concentration and route of administration. Correlation between concentration and depressant effect of alcohol was absent by the po route, and positive by the iv route. When ethanol was administered ip, the depressant effect increased with an increasing concentration up to 70%, but showed a significant reduction at 90%.

95. Aubinière

DE L'IVRESSE D'APRÈS LES BOISSONS INGÉRÉES ET DE SON TRAITEMENT.

[Intoxication after ingested beverages and its treatment].

Journal de la Santé (Paris), 23: 73-76 (0 ref.), 1906.
 F – general – DC (antidotal) – humans – elect., water-bal. agents – gastrointest. agents – *CAAAL-0
 A-0531.

The author discusses the effect of alcohol (loss of appetite, hallucinations, criminality, suicide), beer (indigestion, intestinal troubles, nervous disorders), and wine (alleged to be less dangerous than beer),

and offers treatment for inebriety consisting of a preparation of ammonium acetate (8 grains), sea salt (4 grains), strong coffee (50 grains, liquid), and simple syrup (20 grains). The advocated purgative consists of manna (15 grains), honey (15 grains), senna follicule (10 grains), and sodium sulphate (10 grains), and the suggested emetic is 0.05 centigrams ipecac powder in a glass of water. For best results the author recommends that treatment is to be followed by a relatively moderate and bland diet, but advocates drinking a great deal of water, water combined with milk, and drinks containing lemon juice, light vinegar, or bitters (e.g. gentian bitters).

96. Aufdermaur, M., and Muheim, E.

AKUTE TETRACHLORKOHLSTOFF-VERGIFTUNG IM AKUTEN

ALKOHOLRAUSCH. [Acute carbon tetrachloride poisoning during acute alcohol intoxication].

Z. Unfallmed. Berufskr. (Zurich), 46: 275-282 (22 ref.), 1953.

G – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – absorp., distrib., stor. – liver, kidney – anti-infectants – *CAAAL-6777-E3 A-0532.

A man drank heavily, and was then exposed to the fumes of carbon tetrachloride (CCl_4) for 1 hr. He was admitted to hospital 4 days afterwards with CCl_4 poisoning, and died 11 days later of uremia. Alcohol may precipitate susceptibility to CCl_4 poisoning in two ways: a) in bringing about tissue damage, and b) in potentiating CCl_4 poisoning, because CCl_4 is not soluble in water but easily soluble in alcohol. Furthermore, a liver already damaged by chronic alcohol consumption may add to the dysfunction of the kidneys.

97. Backhouse, C. I., and James, I. P.

THE RELATIONSHIP AND PREVALENCE OF SMOKING, DRINKING AND DRUG TAKING IN (DELINQUENT) ADOLESCENT BOYS.

Brit. J. Addict. (London), 64: 75-79 (6 ref.), 1969.

E – SEC – stat. surv. – conj. addict. – drug-dep. humans – *CAAAL-13719 B-0540.

83% (241) of 290 14-16 year-old boys interviewed at an English detention centre (1967) admitted smoking tobacco—61% being moderate smokers (less than 20 cigarettes/day) and 22% heavy smokers (20 or more cigarettes/day). Generally, the earlier the age at which smoking commenced, the higher was adolescent cigarette consumption. A positive correlation existed between smoking, drinking, and drug-taking. Only 22% of non-smokers drank alcohol regularly, and 4% had tried drugs, as compared with 28% and 10% respectively for moderate smokers, and 37% and 17% respectively for heavy smokers. 37% (107) of the boys denied drinking alcohol; 29% (84) were occasional drinkers (less than once a week); 27% (79) were “regular” drinkers (always once a week and usually every week-end), and 7% (20) drank excessively. 12% (31) of the boys had taken drugs—10 regularly, 21 occasionally, and the majority more than 1 drug. Only 1 boy took drugs before 14 years of age. 24 boys had taken amphetamines, 21 cannabis, 6 barbiturates, 2 heroin, 1 cocaine, and none methylamphetamine. The drinking and drug-taking habits of 14-16 year-old boys were most likely higher in this study than in Britain's general population. The authors feel that a general factor relates drug-dependency habits to delinquency, but the relation between drug use, whether legal or illegal, synthetic or natural, socially acceptable or not, is complex.

98. Baer, G.

BEITRAG ZUR KENNTNISS DER ACUTEN VERGIFTUNG MIT VERSCHIEDENEN ALKOHOLEN. [Contribution to the information on acute poisoning by different alcohols].

Archiv für Anatomie und Physiologie (Leipzig), 22: 283-296 (26 ref.), 1898.

G – exp. comp. – congen. stud. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – mot. perform. – cardiovasc. – G.I. tract – metab. proc. – respir. – skel., muscle, skin – alcohols – *CAAAL-O A-1254.

Rabbits were force-fed various concentrations of methyl, ethyl, propyl, butyl, and amyl alcohol; also ethyl alcohol plus 4%, 2% and 1% solutions of propyl, butyl and amyl alcohol, and ethyl alcohol plus 2% and 1% solutions of furfural. When administered, the alcohol or sol was diluted with water. The symptoms of the rabbit were divided into 3 classes: light, medium, and strong or lethal. From the lethal dosage/kg body weight, the author finds that, relative to ethyl alcohol, methyl, propyl, butyl, and amyl alcohol are, respectively, 0.8, 2.0, 3.0 and 4.0 times as toxic, i.e., the toxicity increases with the boiling point of the alcohol. An addition of 4% of a higher alcohol to ethyl alcohol increased its toxicity noticeably, 2% increased it only slightly, and 1% produced a barely significant increase, the increase being greatest with amyl alcohol and least with propyl alcohol. Furfural increased toxicity even more than amyl alcohol, 1% producing a noticeable increase. The author compares his results with those of others, and concludes that, since the concentrations of (other than ethyl) alcohols occur in much smaller quantities (.3 to .5%) in generally-consumed spirits, the effects of these must be attributed mainly to ethyl alcohol.

99. Ballatore, C.
 L'AZIONE DELLA MORFINA SULL'ALCOOLEMIA PROVOCATA IN SOGGETTI NORMALI. [The effect of morphine on alcoholemia induced in normal patients].
 Boll. Soc. Ital. Biol. Sper. (Naples), 13: 152-153 + 1 table (2 ref.), 1938.
 I – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – dose resp. – blood lev. – CNS – analg., antipyret. – *CAAAL-0 A-1372.

The effects of morphine on the blood alcohol level were investigated in 5 fasted, healthy humans (3 male, 2 female). On one occasion, the subjects were given approximately 2 cc/kg alcohol sc, and, on a second occasion, the same alcohol dose was given 25-35 min after sc administration of 1 cg morphine hydrochloride. It was found that the blood alcohol curves after morphine administration were flatter than after alcohol alone. The peak blood alcohol concentrations in the 5 subjects after morphine were 49%, 41%, 30%, 50%, and 25% lower, respectively. This "hypoalcoholemic" effect of morphine observed confirms the results of Serianni and Baglioni (Accademia Nazionale dei Lincei, Classe di Scienze Fisiche, Matematiche, e Naturali, Rendiconti, 24: 485-487, 1936).

100. Ballatore, C.
 L'AZIONE DELLA MORFINA SULLA CURVA ALCOOLEMICA PROVOCATA IN SOGGETTI AFFETTI DA CIRROSI DEL FEGATO. [The effect of morphine on the blood alcohol curve in patients with cirrhosis of the liver].
 Rivista Ospedaliera (Rome), 31(5-6): 171-181 (8 ref.), 1941.
 I – exp. cont. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – metab. proc. – analg., antipyret. – *CAAAL-4491-A10 A-0533.

Five patients with cirrhosis of the liver received 0.5 cc alcohol/kg po, and their blood alcohol curves were plotted. On another occasion, the patients received 1 cg morphine hydrochloride plus the same alcohol dose. Whereas normal subjects showed a lower blood alcohol curve after administration of morphine (according to previously published studies), three of the cirrhotic patients showed no such lowering; in the other 2 the results were indefinite. It is hypothesized that, while morphine normally stimulates the liver to oxidize alcohol, the cirrhotic liver cannot be so stimulated.

101. Balodis, K.
 WEITERE VERSUCHE ÜBER AUFHEBUNG ÖRTLICHER ANÄSTHESIE DURCH GEWÖHNUNG AN ALKOHOL. [Further experiments on the inhibition of local anesthesia by alcohol].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 173: 589-594 (10 ref.), 1933.
 G – exp. cont. – exp. comp. – DC (decrease) – mammals – chronic admin. – in vivo – acid-base, blood pH, elect. – anesthetics – *CAAAL-2311-D2 A-0534.

In guinea pigs, lid closure reflex was examined before and after chronic alcoholic intoxication with 6 cc of a 25% alcohol sol/kg body wt daily, and after application of local anesthetics, such as eucaine B, tropacocaine, tutocaine, larocaine, pantocaine, and percaine every third day. It was observed that the desensitizing effect of the drugs is diminished, and disappears completely 18 to 96 days after the beginning of alcohol intoxication.

102. Balodis, K.
VERLÄNGERUNG DER MYDRIATISCHEN WIRKUNG DES SUPRARENINS DURCH GEWÖHNUNG AN ALKOHOL. [Prolongation of the mydriatic effect of suprarenin by habituation to alcohol].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 176: 1-7 (4 ref.), 1934.
 G – exp. comp. – DC (decrease) – DC (supra-add. incr.) – mammals – chronic admin. – in vivo – nerv. syst. – autonomic agents – musculoskel. agents – *CAAAL-0 A-0535.

Experiments were performed to study whether chronic alcohol ingestion is capable of changing the effect of suprarenin, physostigmine, or eumydrine on the dilatation of the pupils. Red-eyed guinea pigs received by intubation 6 cc/kg of a 25% alcohol sol daily, and the pupils were measured with a pupillometer. Results showed that habituation to alcohol prolonged the local mydriatic effect of suprarenin. The miotic effect of physostigmine and the mydriatic effect of eumydrine were diminished and prolonged respectively. This effect of alcohol habituation was taken as evidence that the miotic effect is not one of tolerance to poison, but that it is a symptom of chronic poisoning.

103. Balodis, K.
WIEDERHERSTELLUNG DER INFOLGE VON GEWÖHNUNG AN ALKOHOL AUFGEHOBENEN KOKAINANÄSTHESIE DER HORNHAUT DES AUGES DURCH MILCH. [Restoration, by milk, of cocaine eye anesthesia which had been extinguished by habituation to alcohol].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 176: 456-459 (7 ref.), 1934.
 G – exp. – DC (decrease) – mammals – chronic admin. – in vivo – anesthetics – *CAAAL-0 A-0536.

This study attempts to prove that milk is capable of restoring the cocaine anesthesia of the cornea, which was abolished by habituation to alcohol. The corneas of guinea pigs were cocaine-anesthetized for 8-10 min, and then the animals received 6 cc of 25% alcohol/kg daily. After the anesthesia was abolished entirely, 0.5 cc/kg of boiled milk was injected sc. The alcohol intake was not interrupted during this time. The milk injection was found to restore the anesthetic effect of cocaine.

104. Barbillion
NOTE SUR UNE VARIÉTÉ D'ÉRYTHÈME PASSAGER SURVENANT CHEZ LES INDIVIDUS SOUMIS À LA MÉDICATION CHLORALIQUE ET ALCOOLIQUE. [Note on a variety of transitory erythema in individuals under chloral and alcohol medication].
 Archives de Physiologie Normale et Pathologique (Paris), 9: 67-80 (12 ref.), 1887.
 F – general – DC (sensit.) – humans – CNS – skel., muscle, skin – sed., hypnot. – *CAAAL-0 A-0537.

The author points out that individuals subjected to prolonged treatment with chloral are exposed to adverse reactions when ingesting an alcoholic beverage. An example is given in which a child between 4 and 8 yr of age under treatment with chloral (2 to 4 g within a 24 hr period) showed severe erythema 15 to 30 min following ingestion of wine; 5 other similar cases are described in detail. It is pointed out that chloral alone, in a dose of less than 3-4 g, does not ordinarily cause skin eruptions. The author

formulates the opinion that the combination of both substances in question produces eruptions, whereas the individual administration of either substance may produce eruptions only if given in a sufficiently high dose.

105. Barboriak, J. J.

DRUG REACTIONS AFTER INGESTION OF ALCOHOL.

Wisconsin Med. J. (Madison), 63: 213-214 (19 ref.),

1964.

E – general – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – blood lev. – mot. perform. – CNS – metab. proc. – analg., antipyret. – barbiturates – hormones, hormone antag. – tranquilizers – unclass. ther. agents – *CAAAL-0 A-0538.

The literature on the ability of ethanol to produce undesirable reactions when administered with certain drugs is reviewed. Discussed are the side effects and potentiating action of hypoglycemic sulfonylureas, tranquilizers, barbiturates, anticoagulants, and insulin, in combination with alcohol. The severity of reactions after barbiturates seems to be related to the concentration of blood alcohol. The metallic taste of alcohol beverages after sulfonylurea administration suggests the use of these drugs for treatment of chronic alcoholism.

106. Bardoděj, Z., Krivucová, M., and Pokorný, F.

POKUS O BIOCHEMICKÝ VÝKLAD NESNÁŠENÍ ALKOHOLU PŘI INTOXIKACI TRICHLOROETHYLENEM. [A biochemical explanation of alcohol intolerance in trichloroethylene intoxication].

Pracovní Lékařství (Prague), 7: 263-267 (30 ref.),

1955.

C – ES – RS – exp. – DC (sensit.) – humans – blood lev. – cardiovasc. – metab. proc. – respir. – analg., antipyret. – *CAAAL-0 A-0539.

After alcohol administration, an increased acetaldehyde level was found in the blood of humans exposed to trichloroethylene vapours. The intolerance reaction is similar to that after stopetyl (disulfiram); it is due to the inhibition of the oxidation mechanism of the organism by trichloroethylene metabolism, and is characterized by peripheral dilation, fall of blood pressure, tachycardia, tachypnoea, and dyspnoea. The authors consider it to be an acetylcholine reaction. The mechanism probably involves the inhibition of the liver aldehyde dehydrogenase. The alcohol test as a test of physiological functional fitness is recommended for assessing the capacity of employees working with trichloroethylene.

107. Bardoděj, Z., and Vyskočil, J.

THE PROBLEM OF TRICHLOROETHYLENE IN OCCUPATIONAL MEDICINE: TRICHLOROETHYLENE METABOLISM AND ITS EFFECT ON THE NERVOUS SYSTEM EVALUATED AS A MEANS OF HYGIENIC CONTROL.

A.M.A. Archives of Industrial Health (Chicago), 13: 581-592 (30 ref.),

1956.

E – general – DC (sensit.) – humans – cardiovasc. – CNS – metab. proc. – respir. – analg., antipyret. – *CAAAL-0 A-0540.

The literature on the topic is reviewed. Discussed are: trichloroethylene metabolism and its effect on the nervous system, occupational hazards of trichloroethylene and preventive measures, trichloroethylene determination in the atmosphere, trichloroacetic acid determination in the urine, the concentration ratio in urine, the level of atmospheric trichloroethylene, the pathological stages of acute trichloroethylene intoxication, and intolerance to alcohol as the most important symptom.

108. Bardoděj, Z.

INTOLERANCE ALKOHOLU PO CHLORALHYDRÁTU. [Intolerance to alcohol produced by chloral hydrate].

Cesk. Farm. (Prague), 14(9): 478-481 (33 ref.), 1965.
 C – ES – GS – RS – exp. cont. – DC (sensit.) – humans – acute admin. – in vivo – blood lev. – metab. proc. – sed., hypnot. – *CAAAL-11824-B1 B-0246.

Experiments were conducted on 8 volunteers aged 24 to 36. 5 subjects received 1 or 2 g of chloral hydrate for 5 days, and 3 control subjects received no pretreatment. After ingestion of 20 g alcohol, pretreated subjects all experienced a disulfiram-like reaction—4 of the 5 subjects had 105-320 μ g acetaldehyde/100 ml blood. The 3 controls had acetaldehyde levels of 60-75 μ g/100 ml. The authors consider the reaction to be due to changes in acetaldehyde metabolism, but an exhaustive elucidation of the mechanism of the intolerance symptoms cannot be given yet.

109. Barkman, R., and Perman, E. S.
 SUPERSENSITIVITY TO ETHANOL IN RABBITS TREATED WITH COPRINUS
 ATRAMENTARIUS.

Acta Pharmacol. (Copenhagen), 20: 43-46 (10 ref.), 1963.
 E – exp. cont. – DC (sensit.) – mammals – acute admin. – in vivo – dose resp. – cardiovasc. – respir. – *CAAAL-10695-B2 A-1255.

In an experiment to determine if rabbits are rendered supersensitive to ethanol by ingestion of *Coprinus atramentarius*, ethanol (0.2-0.6 g/kg), diluted in saline, was infused iv (1.0 ml/min) 4 hr after pretreatment with 50 or 20 g *C. atramentarius*, 50 g *Psalliota hortensis*, or alone as control. Hypotension, tachycardia and hyperventilation persisted for more than 24 hr after ethanol administration to rabbits pretreated with 50 g *C. atramentarius*, while no corresponding reactions were observed in the other 3 groups. Rabbits are supersensitive to ethanol by moderate amounts of *C. atramentarius*, by disulfiram, and by calcium carbimide, the former 2 substances producing quite similar reactions. Since these 3 agents also induce supersensitivity to ethanol in man, similar mechanisms of action may exist; it is recommended, therefore, that the rabbit should be utilized to isolate and identify the active chemical agent in *C. atramentarius*, which might then be used as an alternative adjunct in the treatment of alcoholism.

110. Barlow, O. W.
 STUDIES ON THE PHARMACOLOGY OF ETHYL ALCOHOL. I. A COMPARATIVE
 STUDY OF THE PHARMACOLOGIC EFFECTS OF GRAIN AND SYNTHETIC ETHYL
 ALCOHOLS. II. A CORRELATION OF THE LOCAL IRRITANT, ANESTHETIC AND
 TOXIC EFFECTS OF THREE POTABLE WHISKEYS WITH THEIR ALCOHOLIC
 CONTENT.

J. Pharmacol. Exp. Ther. (Baltimore), 56(2): 117-146 (22 ref.), 1936.
 E – exp. cont. – exp. comp. – congen. stud. – humans – mammals – other org. – acute admin. – in vivo – in vitro – dose resp. – species or sex diff. – cardiovasc. – CNS – G.I. tract – nerv. syst. – *CAAAL-1784-D2 A-0541.

Experiments were performed with humans, rats, frogs, and other subjects. The presence of congeners in whiskey accentuated the local irritant properties of alcohol. This was shown by a comparison of the effects of whiskey and equivalent concentrations of ethyl alcohol after sc injection into the rabbit ear, and by the greater incidence of emesis and greater degree of gastritis in cats. The toxicity of whiskey for paramoecia clearly exceeded that of equivalent concentrations of pure ethyl alcohol. The results from ip administration in the rat, po and iv administration in the rabbit, and po administration in the cat permit similar conclusions. Different experiments are described and the results presented in detailed tables.

111. Bartlett, G. R.
 INHIBITION OF METHANOL OXIDATION BY ETHANOL IN THE RAT.
 Amer. J. Physiol. (Bethesda), 163: 619-621 (5 ref.), 1950.

E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – in vitro – other drug lev. – liver, kidney – metab. proc. – alcohols – *CAAAL-5754-A2 A-1244.

The inhibition of methanol oxidation by ethanol was investigated in 2 rats and in rat liver slices. 1 rat was given 2 ml of an aqueous sol containing 200 mg of radioactive methanol. After 3 hr, and again after 12 hr, 1 ml of ethanol (50% v/v) was administered po. After ethanol administration, methanol combustion, as measured by the radioactivity of CO₂ respiration, was blocked, and returned to normal in a time corresponding to the metabolic rate of ethanol. The second rat received 1 ml of ethanol (50% v/v) 1/2 hr before, and every 4 hr after, the same dose of methanol. During continuous ethanol feeding, 30% of the administered radioactivity counts were combusted to CO₂, and 24% appeared as methanol in the expired air, compared to 65% in the CO₂ and 15% as methanol in a control rat receiving methanol alone. In rat liver slices, a 0.1 M ethanol sol produced a 72% inhibition of methanol combustion. It is concluded that ethanol produces a very considerable depression of the oxidation of methanol, both in the intact animal and in liver slices.

112. Bartley, A. H.

REMEDY FOR DRUNKENNESS.

Brit. Med. J. (London), 1: 163 (0 ref.),

1953.

E – general – DC (unspec.) – humans – sed., hypnot. – *CAAAL-6478-N4

A-0542.

The author relates his experience with a dozen or so cases of using iv pentothal sodium when confronted with a wildly excited and abusive drunkard. The drug was slowly administered in a dosage of 1 g in 10 ml distilled water, as apnoea may occur if the dose is given too quickly. Immediate immobilization without any side effects was achieved.

113. Bartoníček, V.

DER EINFLUSS EINIGER STOFFE AUF DIE HARNAUSSCHIEDUNG VON TRICHLORÄTHANOL UND TRICHLORESSIGSÄURE BEIM KANINCHEN. [The effect

of various substances on urinary excretion of trichloroethanol and trichloroacetic acid in the rabbit].

Int. Arch. Gewerbepath. (Berlin), 18: 317-326 (19 ref.),

1960.

G – SEC – exp. cont. – exp. comp. – DC (decrease) – DC (sensit.) – mammals – acute admin. – chronic admin. – in vivo – other drug lev. – liver, kidney – anesthetics – *CAAAL-0

A-1256.

6 rabbits were given 1 ml trichloroethylene 3 times weekly. After 7 weeks, while continuing this treatment, ethanol, fructose, glucose, and sodium lactate were separately administered iv. The amount of trichloroethanol and trichloroacetic acid in the urine as a percentage of the amount of administered trichloroethylene was measured. The percentage of trichloroethanol was increased 13% by glucose and 9% by ethanol, and lowered 7% by sodium lactate and 12% by fructose. The intolerance of alcoholics to trichloroethylene is mentioned, as is the use of fructose as therapy for delirium tremens. The percentage of trichloroacetic acid in the urine was unchanged by any of the substances. The amount of eliminated metabolic products was found to be independent of the amount of discharge. The author concludes that iv administration of sodium lactate or fructose may also have a prophylactic effect on humans chronically exposed to trichloroethylene, and suggests similar experiments on human volunteers.

114. Bartoníček, V.

THE EFFECT OF SOME SUBSTANCES ON THE ELIMINATION OF TRICHLOROETHYLENE METABOLITES.

Arch. Int. Pharmacodyn. (Gand), 144(1-2): 69-85 (21 ref.),

1963.

E – exp. cont. – exp. comp. – DC (sensit.) – humans – chronic admin. – in vivo – blood lev. – other drug lev. – acid-base, blood pH, elect. – liver, kidney – metab. proc. – analg., antipyret. – *CAAAL-10630-B1

A-0543.

Eight human volunteers were exposed to trichloroethylene at a concentration of 1042 mg/l of air. The elimination of trichloroethanol (TCE) and trichloroacetic acid (TCAA) in the urine was measured for 22 days. Studied were the effects of glucose, fructose, or sodium lactate given by injection, or of 200 ml of a 25% alcohol sol given po. Alcohol was given before exposure and for 4 days afterwards to subjects. The proportion of trichloroethylene eliminated as TCE and TCAA was 78.1% in controls and 87.5% after alcohol.

115. Baštecký, J.
INKOMPATIBILITA PSYCHOFARMAK. [Incompatibility of psychopharmacological agents].
 Aktiv. Nerv. Sup. (Prague), 8(3): 315-317 (16 ref.), 1966.
 C – SEC – general – DC (add., infra-add., unspec. incr.) – humans – mammals – enzymes –
 tranquilizers – *CAAAL-0 B-0247.

Reviewed is the literature on the incompatibilities of psychopharmacological drugs in clinical experience. The incompatibilities of these drugs, alone and with other drugs or components of food, are discussed. Medical complications and their treatment are outlined. The author notes that chlorpromazine and other neuroleptics prolong alcoholic narcosis and increase the toxicity of alcohol; it can be said that almost all psychotropic drugs are incompatible with alcohol.

116. Bastrup, J. T.
ON THE EXCRETION OF FORMIC ACID IN EXPERIMENTAL POISONING WITH METHYL ALCOHOL.
 Acta Pharmacol. (Copenhagen), 3: 312-322 (11 ref.), 1947.
 E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – CNS – liver, kidney – metab.
 proc. – alcohols – *CAAAL-4871-A2 A-1257.

The effect of simultaneous administration of methyl alcohol (MeOH) and ethyl alcohol (EtOH) on the excretion of formic acid was studied in rabbits and dogs. A rabbit, weighing 2470 g, was administered 6 g MeOH plus 2 g EtOH/kg body wt, followed by 4 g EtOH/kg at 6 hr and 30 hr. On the third day, the rabbit became moribund and died. In 48 hr, 2.9 mg of formic acid were excreted, compared to 214 mg excreted earlier in 44 hr by the same animal after administration of 6 g MeOH alone. A dog, weighing 6.3 kg, was administered 2 g MeOH plus 2 g EtOH, followed by 5.28 g EtOH over 78 1/2 hr. The dog survived the treatment, and in 101 1/2 hr excreted 1834 mg formic acid, compared to 2867 mg excreted earlier in 95 1/2 hr by the same animal after administration of 2 g MeOH alone. It is concluded that, in rabbits and dogs, ethyl alcohol markedly inhibits formic acid excretion after administration of methyl alcohol.

117. Baumann, H.
EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE BEEINFLUSSUNG DER BLUTALKOHOLKURVE UND PSYCHOMOTORIK DURCH KAFFEE. [Experimental investigations on the influence of coffee on the blood alcohol curve and psychomotor performance].
 Dissertation, Medical Faculty of the University of Berne, Switzerland, 44 pp. (24 ref.), 1952.
 G – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – mot. perform. –
 psychol. perform. – stimulants – *CAAAL-0 A-0544.

The Billes Test (hand dexterity), the two-hand test, an acoustic reaction test, the Bourdon test, the tapping test, and the picture description test were applied to 6 human subjects under three conditions; sober, after 1 liter of wine, and after a wine-coffee combination. The blood alcohol content was determined after a modification of Widmark's method. The alcohol-coffee combination was found to improve the performance and decrease the intoxication.

118. Baxter, R. C., and Hensley, W. J.

THE EFFECT OF ETHANOL AND CYANIDE ON NAD-NADH₂ RATIOS IN RAT LIVER.

Biochem. Pharmacol. (New York), 18(1): 233-236 (14 ref.),

1969.

E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – other org. – acute admin. – in vivo – metab. proc. – miscellaneous – *CAAAL-13990 B-0501.

45 min after administration via stomach tube of 2 ml water (control), 25% ethanol (2.5 g/kg) (ethanol-fed), 2 mM potassium cyanide (1.25 mg/kg) (cyanide-fed), or ethanol plus cyanide in 2 ml water (ethanol- and cyanide-fed), 4 groups of male albino rats, 7-8 weeks old and weighing 200-250 g, were killed and the livers removed and processed for determination of concentration of lactate, pyruvate, glutamate, 2-oxoglutarate and ammonia. Using substrate and coenzyme ratios to determine both cytoplasmic and mitochondrial NAD/NADH₂ ratios, the results indicated, as expected, that ethanol decreased both ratios; that cyanide, by inhibiting electron transport, decreased the mitochondrial NAD/NADH₂ ratio; and that ethanol plus cyanide decreased the mitochondrial ratio even further. Unexpected, however, was the 2-fold increase in cytoplasmic ratio and the reduction of pyruvate concentrations in the cyanide-fed livers. The ethanol-cyanide combination reduced the cytoplasmic ratio somewhat, mainly because of ethanol oxidation (i.e., production of NADH₂). The cyanide-increased cytoplasmic ratio finding leads to the postulation of a non-enzymatic cyanide-catalysed conversion of methylglyoxal to pyruvate, which is then converted to lactate with the production of NAD (which is responsible for the ratio increase).

119. Beck, W. V.

BEEINFLUSSUNG DES BLUTALKOHOLSPIEGELS BEI VERBRENNUNG UND EINATMUNG VON BRANDGASEN. (EXPERIMENTELLE UNTERSUCHUNGEN.) [The

influence on the blood alcohol level of combustion and inhalation of fumes emanating from a fire. (Experimental investigations)].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 33: 95-102 (4 ref.),

1940.

G – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – indust. intox. – *CAAAL-0 A-0545.

Tests on rabbits to determine whether carbon monoxide dissolved more quickly in alcoholized blood proved negative. Both the control animal and the one administered 4 g alcohol died 17 min after exposure to the gas. It is therefore concluded that death results through carbon monoxide poisoning before any other substance may take effect.

120. Beckman, W. W., Aub, J. C., Mallory, T. B., and Kiyasu, W.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL; WEEKLY CLINICOPATHOLOGICAL EXERCISES: CASE 34221.

New Eng. J. Med. (Boston), 238(22): 776-779 (1 ref.),

1948.

E – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – humans – other drug lev. – acid-base, blood pH, elect. – blood comp., sites, lymph – CNS – liver, kidney – anti-infectants – *CAAAL-0 A-0546.

A case of fatal carbon tetrachloride poisoning is described in detail and commented upon in a seminar-type situation. The initial clinical diagnosis was acute nephritis, but the autopsy and later examinations of the evidence pointed to carbon tetrachloride poisoning with lower nephron nephrosis, acute central necrosis of the liver, pulmonary atelectasis, and moderate arteriosclerosis. It is suspected that the patient had already consumed a fair amount of alcohol, and that his alcoholic habits enhanced the poisoning.

121. Bedaux, F. C.
 GENEESMIDDELEN, ALCOHOL EN VERKEER. [Drugs, alcohol and traffic].
 Pharm. Weekbl. (The Hague), 102: 990-1001 (43 ref.), 1967.
 D – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unspec.) – humans – blood
 lev. – metab. proc. – analg., antipyret. – anti-infectants – barbiturates – hormones, hormone antag.
 – unclass. ther. agents – *CAAAL-0 B-0248.

The author refers to literature on the use of alcohol and other drugs by motorists, and the frequent combination of both. Of 5,000 drivers with a blood alcohol level of about 1.5‰, 16% combined alcohol and drugs in doses considerably higher than drug doses used by the non-drinking drivers. Details of the interaction mechanisms are scant. Basically, three types of actions are distinguished: a) drugs which alter the action of alcohol, b) those which reduce its effect, and c) those which increase it. Examples of drugs belonging to the above groups are cited: disulfiram, metronidazole (flagyl), thiocyanates, and *Coprinus atramentarius* are included in the a) group; the b) group includes insulin, dinitrophenol and tri-iodothyronine; and for c), groups of drugs including analgesics and barbiturates. Drug groups indexed in the K.N.M.P. Documentation System which exercise a synergistic effect in combination with alcohol are tabulated. 20 groups of drugs and the degrees of synergism are classified, including chemotherapeutics.

122. Beer, C. T., and Quastel, J. H.
 THE EFFECTS OF ALIPHATIC ALCOHOLS ON THE RESPIRATION OF RAT BRAIN
 CORTEX SLICES AND RAT BRAIN MITOCHONDRIA.
 Canad. J. Biochem. (Ottawa), 36: 543-556 (14 ref.), 1958.
 E – exp. cont. – DC (decrease) – mammals – acute admin. – in vitro – CNS – respir. – elect., water-bal.
 agents – *CAAAL-8725-B2 A-1258.

Experiments were carried out to study the effects of aliphatic alcohols (n-propanol, n-butanol, n-pentanol, and ethanol) on the respiration of rat brain cortex slices and mitochondria, both in normal glucose-Ringer media and in media with added potassium ions to stimulate respiration. 50-70 mg brain slices of female hooded rats were used, and the experiments were performed using the Warburg manometric apparatus at 37° C. The alcohols were predistilled to eliminate contamination. The results showed that ethanol increased the rate of oxygen consumption of the unstimulated brain respiration, and decreased the rate of potassium ion-stimulated brain cortex respiration by 52%. When using higher aliphatic alcohols, a slight drop in the unstimulated respiratory rate was observed. However, the potassium ions made the tissues so sensitive to the aliphatic alcohols that an even lower rate of respiration than the unstimulated was observed. The addition of ethanol to the brain mitochondria did not significantly alter the respiratory rate. The results were tabulated, compared, and graphically evaluated. The authors conclude that ethanol and higher aliphatic alcohols have little or no effect on unstimulated brain respiration, but have marked effects on stimulated respiration. These effects are most probably due to blocking of the entry of substrates into the stimulated brain cells by the alcohols.

123. Behr, A.
 BEITRAG ZUR CASUISTIK DER PARALDEHYDDELIRIEN UND BEMERKUNGEN
 ÜBER DIE TRUNKSUCHT DER FRAUEN BESSERER STÄNDE. [Contribution to the
 casuistics of paraldehyde deliria, and remarks about the alcoholism of women of the better
 classes].
 St. Petersburg Medicinische Wochenschrift (St. Petersburg), 27: 127-131 (15 ref.), 1902.
 G – SEC – general – conj. addict. – drug-dep. humans – CNS – analg., antipyret. – *CAAAL-0
 A-0547.

The author, a physician, discusses the abuse of drugs, and cites a case history of a woman who was given a prescription of paraldehyde. She became an addict, starting with a tablespoonful and culminating in 15.0 g/day. After a few months of drug intake, the woman's weight was down to 81 pounds

and she was hospitalized. The patient was suffering from hallucinations and tremor. The author points out that symptoms like delirium tremens in morphine addicts were often finally diagnosed as being caused by simultaneous alcohol ingestion, but, in the reported case, the symptoms were indubitably caused by paraldehyde.

124. Benassi, G.
UN CASO DI AVVELENAMENTO MORTALE NELLA PREPARAZIONE DEL PIOMBO TETRAETILE. [A case of fatal poisoning from tetraethyl lead preparation].
Rassegna di Medicina Industriale e di Igiene del Lavoro (Turin), 10: 390-398 (3 ref.), 1939.
I – SEC – general – case hist. – post-mort. – humans – blood lev. – other drug lev. – miscellaneous
– *CAAAL-0 A-1373.

A case history is reported of fatal lead poisoning in a 47 yr-old former alcoholic. After 4 months of work involving exposure to tetraethyl lead, the patient began to feel nausea and vague gastric disorders, with a general weak feeling, followed within a few days (Sept. 15) by trembling, an increase of pre-existent stuttering, and anxiety. When examined on Sept. 19, he exhibited, in addition, a mildly delirious state at intervals, with some hallucinations. After being admitted to hospital on Sept. 20, the agitation kept increasing until restraint was necessary, and the delirium became continuous, with more frequent hallucinations. On Sept. 22, the patient lapsed into a coma and died within 15 min. The autopsy revealed very few slight abnormalities. Traces of lead were found in the parts tested (brain, liver, kidneys, urine), but this was not sufficient to warrant a poisonous cause of death, since the body can retain a certain amount of lead without succumbing. On the other hand, in the period of about 15 days between the last working day and death, a good portion of the poison could have been eliminated. That the previous alcoholism might have been a joint causal factor is suspected, although there were no evident and permanent organic alterations.

125. Bennett, I. L., Jr., Cary, F. H., Mitchell, G. L., Jr., and Cooper, M. N.
ACUTE METHYL ALCOHOL POISONING: A REVIEW BASED ON EXPERIENCES IN AN OUTBREAK OF 323 CASES.
Medicine (Baltimore), 32: 431-463 (116 ref.), 1953.
E – review – DC (antidotal) – humans – mammals – in vivo – in vitro – blood lev. – other drug lev.
– absorp., distrib., stor. – acid-base, blood pH, elect. – metab. proc. – alcohols – *CAAAL-6851-E4
A-0548.

A review of publications on methanol poisoning and its treatment is presented. Clinical material, chemistry and pharmacology, symptoms, and treatment are described in detail. The relationship between the metabolism of ethyl and methyl alcohol has not been decided; blood ethanol levels in the fatal cases were usually higher than blood methanol. More experiments are needed before ethanol is used as a treatment; its substitution for alkalization is not recommended. The “relatively innocuous sequelae” of overdosage with bicarbonate in the experience of the authors leads them to disagree with Røe (Acta Med. Scand. (Stockholm), 126(Suppl. 182): 253 pp., 1946), who advocates the use of ethyl alcohol in suspected methanol poisonings, until arrangements can be made for determination of plasma bicarbonate prior to use of alkali.

126. Berger, H. J.
CHLORPROMAZINE AND ETHANOL COMBINATION: EFFECTS ON RESPIRATION, RANDOM MOTOR ACTIVITY AND CONDITIONED AVOIDANCE-ESCAPE IN MICE.
Quart. J. Stud. Alcohol (New Haven), 30(4A): 862-869 (14 ref.), 1969.
E – exp. cont. – DC (supra-add. incr.) – mammals – in vivo – mot. perform. – CNS – nerv. syst. – respir. – skel., muscle, skin – tranquilizers – *CAAAL-12722-D2 B-0494.

Groups of 4 male Swiss-Webster mice (20-28 g) were given 2 sc injections, 15 min apart, of: 1) saline-saline (0.3 ml), 2) chlorpromazine (CPZ—1.25 or 2.5 mg/kg)-saline, 3) ethanol (0.19, 0.38 and

0.79 mg/kg)-saline, and 4) CPZ-ethanol. The mean random motor activity test results were: 110 counts (number of times light beam broken in photocell activity cage within 30 min) after saline, 18 counts after CPZ (2.5 mg/kg), 86 counts after ethanol (0.38 mg/kg), and 6 counts after CPZ (2.5 mg/kg) plus ethanol (0.38 mg/kg). The CPZ potency was too great in this experiment to demonstrate a possible synergistic effect with alcohol. The mean escape time (seconds to climb a pole after hearing buzzer to avoid shock) results were: 0 sec 25 min after saline (control), 5 sec after CPZ (1.25 mg/kg), 3 sec after ethanol (0.38 mg/kg), and 22 sec after CPZ-ethanol, thus demonstrating a significantly greater combined effect in contrast to the maximal theoretical additive effect of 8 sec. This combined effect is even greater for higher levels of ethanol. The supra-additive action of CPZ and ethanol was also illustrated in an experiment involving depression of the respiratory rate.

127. Berger, H.
 ADDICTION TO METHYPRYLON: REPORT OF CASE OF 24-YEAR-OLD NURSE WITH POSSIBLE SYNERGISM WITH PHENOTHIAZINE.
 J.A.M.A. (Chicago), 177(1): 63-65 (11 ref.), 1961.
 E – SEC – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – drug-dep. humans – cardiovasc. – CNS – liver, kidney – nerv. syst. – sed., hypnot. – tranquilizers – *CAAAL-0
 A-1259.

The case history of a 24-year-old nurse addicted to methyprylon is presented. To terminate a 5 yr barbiturate addiction, she switched to methyprylon, and for 18 months was taking 7,500-12,000 mg/day. She smoked 2-3 packs of cigarettes/day, denied the excessive use of alcohol, and did not take narcotics. She attempted to break her habit under the supervision of a physician who prescribed 0.005 mg methadone, 0.005 mg prochlorperazine, 0.050 mg promazine hydrochloride, and 0.050 mg thiamine chloride to aid withdrawal. Withdrawal symptoms experienced included auditory hallucinations, convulsions, and nervous irritability. After 5 days, the patient died in congestive failure of myocardial degeneration. Autopsy revealed traces of ethyl alcohol in the stomach and promazine in the stomach and brain. It is concluded that the patient died during methyprylon withdrawal, and that alcohol, and promazine, and other substances (e.g. alcohol) used to control withdrawal symptoms, may have contributed to her death. The author does not, however, consider the possibility of a lethal synergism of methyprylon and alcohol.

128. Beringer, A.
 DIE BEHANDLUNG DER ZUCKERKRANKHEIT MIT SB 1. [The treatment of diabetes with SB 1].
 Wien. Med. Wschr. (Vienna), 110: 109-111 (1 ref.), 1960.
 G – SEC – exp. – DC (sensit.) – humans – blood lev. – glands – hormones, hormone antag. – *CAAAL-0
 A-1260.

A 3 month-long treatment of diabetics with a new oral antidiabetic, SB 1, is reported. 75 older diabetics, who had not been previously treated with insulin or another anti-diabetic, received 1 g (2 tablets) of SB 1 each day. When the results were unsatisfactory, the dosage was increased to 3 tablets daily. Examinations were performed on the patients at 2 week intervals. In 57 of the 75 patients, a satisfactory decrease of blood and urea sugar was observed. 12 patients failed to respond—8 of these were successfully treated later with another kind of oral antidiabetic. Disturbing side effects appeared in 5 patients; 3 felt a tension in the gastric region, but the effect disappeared with continued treatment. 1 of the patients developed an intolerance towards alcohol. The author concludes that the substance is successful in treatment of diabetes, although the time of experimental treatment was short and the number of patients too small. The observed side effects would have to be also extensively investigated.

129. Berndt, H., and Kutschke, I.
 DER EINFLUSS VON THIAMINTETRAHYDROFURFURYLDISULFID (TTFD) AUF DEN ALKOHOLABBAU. [The influence of thiamine tetrahydrofurfuryl disulfide (TTFD) on

the metabolism of alcohol].

Klin. Wschr. (Berlin), 45: 685-686 (4 ref.),

1967.

G – ES – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – chronic admin.

– in vivo – blood lev. – metab. proc. – *CAAAL-0

B-0912.

The effect of thiamine tetrahydrofurfuryl disulfide (TTFD) on alcohol metabolism was investigated in 6 men and 3 women. On one occasion, 0.5 g/kg ethanol in the form of 40% vodka was administered po on an empty stomach, and, on a second occasion, the same alcohol dose was given following administration for 7 days of 150 mg TTFD/day po (3 doses/day). Blood alcohol levels were determined immediately before, as well as 30, 60, 90, 150, 210, and 270 min after alcohol administration. Blood alcohol levels 150 min after alcohol were found to be significantly higher following TTFD, while, at 30, 60, 90, 210, and 270 min, these values showed no variation from the preliminary alcohol test. It is concluded that TTFD has a weak inhibiting effect on alcohol dehydrogenase, causing a very slight delay of alcohol metabolism in humans. This effect, however, has no practical significance—therapy with TTFD is not thereby confined, and there are no consequences concerning forensic evaluation of blood alcohol concentration in drivers being so treated.

130. Bernstein, M. E., Richards, A. B., Hughes, F. W., and Forney, R. B.
OPTOKINETIC NYSTAGMUS UNDER THE INFLUENCE OF D-AMPHETAMINE AND ALCOHOL.
In: Harger, Rolla N., ed. *Alcohol and Traffic Safety*. Proceedings of the Fourth International Conference on Alcohol and Traffic Safety at Indiana University, December 6-10, 1965. Bloomington, Indiana: Indiana University Press, pp. 208-210 (0 ref.), 1966.
E – presentation – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – CNS – nerv. syst. – amphetamines – *CAAAL-0 B-0249.

The effect of amphetamine-alcohol combinations on horizontal optokinetic nystagmus was studied in 8 healthy humans. The mean blood alcohol concentrations of the subjects were similar, being 46 mg% in the placebo-alcohol combination and 51 mg% in the amphetamine-alcohol combination. Amphetamine markedly overcame the nystagmatic effects resulting from the ingestion of a moderate amount of alcohol.

131. Bernstein, M. E., Hughes, F. W., and Forney, R. B.
THE INFLUENCE OF A NEW CHLORDIAZEPOXIDE ANALOGUE ON HUMAN MENTAL AND MOTOR PERFORMANCE.
J. Clin. Pharmacol. (New York), 7(6): 330-335 (6 ref.), 1967.
E – exp. cont. – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – *CAAAL-0 B-0250.

Ro 5-4556, a chlordiazepoxide analogue, was tested on motor and mental performance, with and without ethanol, on human volunteers under four double-blind and randomized treatment conditions. Testing was biphasic, with mental performance being analyzed by means of a delayed audio feedback system (DAF), and motor performance by a pursuit meter apparatus. Results indicate that Ro 5-4556, in general, did not affect human mental or motor performance, either alone or in combination with alcohol.

132. Bertram, F., Bendfeldt, E., and Otto, H.
INDIKATIONEN UND ERFOLGE DER PERORALEN BEHANDLUNG DES DIABETES MELLITUS MIT EINEM SULFONYLHARNSTOFFDERIVAT: BERICHT ÜBER 335 FÄLLE. [Indications and success of peroral treatment of diabetes mellitus with a sulfonylurea derivative: report on 335 cases].
Deutsch. Med. Wschr. (Stuttgart), 81: 274-278 (12 ref.), 1956.

G – SEC – general – DC (sensit.) – humans – cardiovasc. – glands – hormones, hormone antag. –
*CAAAL-0 A-1261.

The peroral treatment of diabetes mellitus with a sulfonylurea derivative, nadisan, proved to be successful. Most of the patients showed tolerance towards nadisan, even when given the drug in considerably high doses for a long period of time. However, side-effects did appear in a certain percentage of patients; these effects included gastric hyperacidity, dermatitis, eczema, and a decreased tolerance to alcohol. The percentage of such side-effects, however, was small enough for the drug to be considered a useful therapeutic agent.

133. Bester, J. F.
POTENTIATION OF DRUGS BY ETHYL ALCOHOL.
Amer. Ass. Industr. Nurses J. (Pitman, N.J.), 15(8): 10-12 (11 ref.), 1967.
E – general – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – blood lev. – CNS – metab. proc. – analg., antipyret. – anti-infectants – barbiturates – cardiovasc. agents – enzymes – hormones, hormone antag. – sed., hypnot. – tranquilizers – unclass. ther. agents – *CAAAL-0 B-0251.

Reviewed are the major biological effects of alcohol on the CNS, the liver, respiration, the kidneys, the adrenals, the cardio-vascular system, and the gastro-intestinal system. Also discussed are the interactions of alcohol with the phenothiazines, the barbiturates, the analgesic drugs, disulfiram, the anti-histamines, the sulfonamides, the monoamine oxidase inhibitors, and other drugs.

134. Bethune, H. C., Burrell, R. H., Culpan, R. H., and Ogg, G. J.
VASCULAR CRISES ASSOCIATED WITH MONOAMINE-OXIDASE INHIBITORS.
Amer. J. Psychiat. (Hanover, N.H.), 121: 245-248 (23 ref.), 1964.
E – general – DC (add., infra-add., unspec. incr.) – humans – cardiovasc. – enzymes – *CAAAL-0 A-0549.

8.4% of 692 patients receiving monoamine oxidase (MAO) inhibitors experienced acute hypertensive crises, featured by intense occipital headache and palpitation. When the patients were advised not to eat cheese, the incidence of acute reactions fell to 3.3%. The 8 reactions in the latter series followed the ingestion of cheese, large quantities of cream, or alcohol. The highest incidence of these reactions occurred with tranlycypromine. The combined use of MAO inhibitors and cheese, cream, or alcohol is to be carefully avoided.

135. Betlheim, S., Peršić, N., and Blažević, D.
O DJELOVANJU BARBITURATA NA KRONIČNE ALKOHOLIKE. [The action of barbiturates on chronic alcoholics].
Acta Med. Jugosl. (Belgrade), 8: 68-81 (17 ref.), 1954.
Se – exp. – cross-tol. – drug-dep. humans – acute admin. – in vivo – psychol. perform. – CNS – sed., hypnot. – *CAAAL-6936-I33 A-1262.

The use of hexobarbital in the treatment of chronic alcoholism was evaluated in 20 alcoholics. Each subject was examined for psychopathological phenomena before and after the gradual administration of a subnarcotic dose of hexobarbital (10% sol). It was observed that, after hexobarbital administration, the subjects manifested intensified tremors and amorphous behavior. Confabulation, sensory illusions (mostly optical), spontaneous delirium, as well as delirium induced by suggestive questioning, were among the psychopathological phenomena observed. It was noted that the hexobarbital delirium was similar to the delirium tremens of acute alcoholism, but with less spontaneity in the subjects' movements and sensory illusions, more ambivalence, greater suggestibility and shorter duration. It is concluded that hexobarbital administered to alcoholic subjects produces barbiturate poisoning and

a delirium state similar to delirium tremens. It is recommended, therefore, that barbiturate therapy not be used in treating chronic alcoholics.

136. Biehl, B., Fuhrmann, J., and Seydel, U.
 AUSWIRKUNGEN DER GLEICHZEITIGEN EINNAHME VON ALKOHOL UND
 VITAMINHALTIGEN FRUCHTSÄFTEN AUF PSYCHOLOGISCHE TESTLEISTUNGEN
 UND DIE BLUTALKOHOLKONZENTRATION. [Effects of simultaneous intake of alcohol
 and fruit juices containing vitamins on psychological test results and the blood alcohol
 concentration].
 Z. Exp. Angew. Psychol. (Göttingen), 16(3):402-419 (15 ref.), 1969.
 G – ES – FS – exp. cont. – exp. comp. – DC (decrease) – humans – acute admin. – in vivo – blood
 lev. – mot. perform. – psychol. perform. – absorp., distrib., stor. – CNS – nutritive agents –
 *CAAAL-14651 B-1005.

The effects of alcohol, alone and in combination with fruit juice or a placebo, on blood alcohol concentrations (BAC) and psychomotor performance, were investigated in humans. In 1 experimental series, 120 male students were divided into 3 groups of 40 subjects each. The first group received alcohol placebo + fruit juice placebo, the second group alcohol + fruit juice placebo, and the third alcohol + fruit juice. In a second series, 16 male students received on 1 occasion, alcohol alone. On a second occasion, the same amount of alcohol was given in fruit juice. In both series, all drinks were given in a vol of 2.7 ml/kg, the alcohol consisting of 42% tequila, and corresponding to 0.9 g alcohol/kg. In both series, BAC determinations and psychomotor tests were made 90, 135, and 180 min after alcohol ingestion. Both the BAC (ranging from 0.8 to 1.2°/oo) and the degree of psychomotor impairment were highest after alcohol + placebo, and lowest after alcohol + fruit juice. Significant psychomotor impairment occurred at all test intervals; differences were found between the 2 alcohol groups—those receiving alcohol + fruit juice were significantly less impaired than those receiving alcohol only. Also reported, in more condensed form, in *Alkohol und Verkehrssicherheit*, pp. I.57-I.64, 1969.

137. Biehl, B., Fuhrmann, J., and Seydel, U.
 AUSWIRKUNGEN DER GLEICHZEITIGEN EINNAHME VON ALKOHOL UND
 VITAMINHALTIGEN FRUCHTSÄFTEN AUF PSYCHOLOGISCHE TESTLEISTUNGEN
 UND DIE BLUTALKOHOLKONZENTRATION. [Effects of simultaneous intake of alcohol
 and fruit juices containing vitamins on psychological test results and the blood alcohol
 concentration].
 In: *Alkohol und Verkehrssicherheit*: Konferenzbericht der 5. Internationalen Konferenz über Alkohol
 und Verkehrssicherheit. [Alcohol and traffic safety: proceedings of the 5th International Conference
 on Alcohol and Traffic Safety]. Freiburg im Breisgau, West Germany, 1969. Freiburg im Breisgau:
 Hans Ferdinand Schulz Verlag, pp. I.57-I.64 (3 ref.) @ 1969.
 G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo
 – blood lev. – mot. perform. – psychol. perform. – absorp., distrib., stor. – CNS – nutritive agents
 – *CAAAL-0 B-1006.

The effects of alcohol, alone and in combination with fruit juice or a placebo, on blood alcohol concentrations (BAC) and psychomotor performance were investigated in humans. In 1 experimental series, 120 male students were divided into 3 groups of 40 subjects each. The first group received alcohol placebo + fruit juice placebo, the second group alcohol + fruit juice placebo, and the third alcohol + fruit juice. In a second series, 16 male students received, on 1 occasion, alcohol alone. On a second occasion, the same amount of alcohol was given in fruit juice. In both series, all drinks were given in a vol of 2.7 ml/kg, the alcohol consisting of 42% tequila, and corresponding to 0.9 g alcohol/kg. In both series, BAC determinations and psychomotor tests were made 90, 135, and 180 min after alcohol ingestion. Both the BAC (ranging from 0.8 to 1.2°/oo) and the degree of psychomotor impairment were highest after alcohol + placebo, and lowest after alcohol + fruit juice. Signifi-

cant psychomotor impairment occurred at all test intervals; differences were found between the 2 alcohol groups—those receiving alcohol + fruit juice were significantly less impaired than those receiving alcohol only. Also reported, in expanded form, in *Z. Exp. Angew. Psychol.*, 16(3): 402-419, 1969.

138. Bills, C. E.
A PHARMACOLOGICAL COMPARISON OF SIX ALCOHOLS, SINGLY AND IN
ADMIXTURE, ON PARAMECIUM.
J. Pharmacol. Exp. Ther. (Baltimore), 22(1): 49-57 (6 ref.), 1923.
E – exp. cont. – DC (decrease) – other org. – in vivo – alcohols – *CAAAL-0 A-0550.

The toxic and narcotic effects of methyl, ethyl, n-propyl, i-propyl, n-butyl, and i-butyl alcohols were determined using the simple protoplasmic organism, paramecium. It was found that, from methyl to butyl alcohol, the toxicity steadily increases, and the ratio of narcotic and toxic concentrations reaches a maximum in propyl alcohol. The alcohols are toxic antagonists, but it remains to be determined if the narcotic effect is also antagonized.

139. Binswanger, H., and Kunz, H.
11-DESOXO-CORTISON-ACETAT (SUBSTANZ-S-ACETAT NACH REICHSTEIN) BEI
INTOXIKATIONSZUSTÄNDEN, SPEZIELL BEIM ALKOHOLISMUS.
[11-desoxo-cortisone-acetate (substance S-acetate, according to Reichstein) in toxic states,
especially in alcoholism].
Schweiz. Med. Wschr. (Basel), 81(14): 338-339 (12 ref.), 1951.
G – general – DC (antidotal) – drug-dep. humans – acute admin. – in vivo – mot. perform. – psychol.
perform. – blood comp., sites, lymph – CNS – G.I. tract – liver, kidney – analg., antipyret. – nutritive
agents – *CAAAL-5785-C6 A-0551.

Seven patients in alcoholic toxic states received 60 mg of substance-S-acetate im and 1 g redoxon (an ascorbic acid preparation) iv. The clinical improvement was significant, the treatment resulting in sedation, reduction of tremor, and reappearance of appetite. A hypothesis explaining the action of these drugs is offered.

140. Binz, C.
BEITRÄGE ZUR KENNTNISS DER KAFFEEBESTANDTHEILE. [Contributions to the
knowledge of coffee constituents].
Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 9: 31-51
(49 ref.), 1878.
G – exp. comp. – general – case hist. – DC (decrease) – drug-dep. humans – mammals – acute admin.
– in vivo – mot. perform. – absorp., distrib., stor. – cardiovasc. – CNS – G.I. tract – metab. proc.
– nerv. syst. – respir. – skel., muscle, skin – stimulants – *CAAAL-0 A-0552.

Some 19th century literature on the pharmacology of caffeine is reviewed. Animal experiments and two cases of human caffeine abuse are described and discussed. The author's conclusion is that caffeine and caffeon (the roasted aromatic ingredients of coffee) in average doses have an excitatory effect on brain, heart, respiration and body temperature. In one experiment, caffeine was found to have an antagonistic effect on alcohol narcosis.

141. Binz, C.
UEBER DEN ALKOHOL ALS ARZNEIMITTEL GEMÄSS DEN ERGEBNISSEN DER
FORSCHUNGEN DES LETZTEN JAHRZEHNIS. [Alcohol as medicine, according to the
results of investigations during the last decade].

Berliner Klinische Wochenschrift (Berlin), 40(3): 45-48 (0 ref.), 1903.
 G – exp. – congen. stud. – humans – mammals – acute admin. – in vivo – cardiovasc. – CNS – respir.
 – *CAAAL-0 A-0553.

The author refers to the definition of alcohol as a stimulant, in relation to the encephalon and its psychic functions. Tests on animals given alcohol, and on volunteers given 75 cc sherry, showed that respiratory activity was stimulated to a very considerable extent. Moreover, it is stated that the presence of aromatic components in the alcohol increase the stimulating effect of pure alcohol. Basing his findings on 131 tests on humans, the author emphasizes the therapeutic value of moderate alcohol doses in respiratory and circulatory treatment.

142. Biondi, C.
 L'AZIONE DELL'ALCOOL NEGLI AVVELENATI CRONICI PER PIOMBO, PER MERCURIO E PER ANTIMONIO. [The action of alcohol in chronic poisoning of lead, mercury and antimony].
 Riv. Crit. Clin. Med. (Florence), 7: 584-586 (2 ref.), 1906.
 I – exp. cont. – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – mammals – chronic admin. – in vivo – CNS – miscellaneous – unclass. ther. agents – *CAAAL-0 A-0554.

Upon investigation, the author found that a small quantity of alcohol quickly intoxicated workers in lead, antimony, and mercury mines. Also, smoking after a small drink increased intoxication. Clinical observations of rabbits, treated with a daily dose of 15 to 30 cg lead acetate or carbonate for 3 months, followed by 4.5 to 5 cc alcohol/kg, revealed appreciable narcosis which was absent in the control group. It is concluded that the metallic substances in question markedly reduce the tolerance to alcohol in men and animals.

143. Bisset, G. W., and Walker, J. M.
 THE EFFECTS OF NICOTINE, HEXAMETHONIUM AND ETHANOL ON THE SECRETION OF THE ANTIDIURETIC AND OXYTIC HORMONES OF THE RAT.
 Brit. J. Pharmacol. (London), 12: 461-467 (19 ref.), 1957.
 E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – blood comp., sites, lymph – glands – liver, kidney – hormones, hormone antag. – *CAAAL-8514-D2 A-1374.

Rats received 5 ml/100 g of tap water by stomach tube, followed, in some cases, by pretreatment with 0.5 or 5.0 mg/100 g hexamethonium bromide ip. Nicotine hydrogen tartrate (0.5 or 0.75 mg/100 g sc), pituitrin (6.0 mU/100 g sc or ip) or saline were then administered. In a second experiment, 5 ml/100 g 10% ethanol po was substituted for the water load, followed by the above nicotine or pituitrin doses. It was found that nicotine produced a profound antidiuresis and chloruresis. The previous injection of hexamethonium or administration of ethanol did not inhibit nicotine antidiuresis, but did inhibit the chloruresis. Although not itself an antidiuretic, ethanol increased the mean time to maximal rate of excretion after nicotine from 146 to 187 min; it reduced total chloride excretion from 200 to 149 $\mu\text{g eq Cl}/100\text{ g}$. Since neither hexamethonium nor ethanol affected the chloruretic action of pituitrin, the results suggested the possibility that they inhibit centrally the release of oxytocin, but not of ADH. Male rats (200-300 g) were anesthetized with 5 ml/100 g of 15-20% ethanol (v/v) po, supplemented, if necessary, by 1-4 ml 20% ethanol ip. After anesthesia had been obtained, hexamethonium or nicotine was injected, and venous blood collected for oxytocin assay. Nicotine caused a release of oxytocin into the blood which was not blocked by ethanol, nor significantly reduced by hexamethonium.

144. Bjerver, K., and Goldberg, L.
 ALCOHOL TOLERANCE IN INDIVIDUALS WITH CHRONIC PRODUCER-GAS INTOXICATION.

Quart. J. Stud. Alcohol (New Haven), 9(3): 329-352 (11 ref.), 1948.
 E – exp. cont. – DC (unchanged) – humans – acute admin. – chronic admin. – in vivo – blood lev.
 – mot. perform. – psychol. perform. – absorp., distrib., stor. – CNS – senses – alcohols – indust. intox.
 – *CAAAL-4144-A1 A-0555.

The alcohol tolerance in 11 patients with chronic producer-gas (containing 20-30% carbon monoxide) intoxication was established quantitatively by a number of tests in relation to the blood alcohol curve. The results were compared with those in a control group of 7 healthy subjects. Alcohol tolerance in the experimental group was the same as in the controls. No difference was found between the former and the latter with respect to the blood alcohol curve.

145. Björnström, F.
 OM SPRITDRYCKERS VERKAN VID SAMTIDIGT BRUK AF CHLORALHYDRAT. [On the effect of alcoholic beverages and simultaneous use of chloral hydrate].
 Upsala Läkareförening, Förhandlingar (Stockholm), 8: 114-116 (0 ref.), 1872.
 S – general – DC (sensit.) – humans – cardiovasc. – analg., antipyret. – sed. hypnot. – *CAAAL-0 A-0556.

Cases are cited of a 22 yr-old male and a 68 yr-old female treated for melancholia with chloral hydrate in doses of 3 g/day and 1 g/2 days, respectively. In each case, the ingestion of 1/2 bottle of beer at mealtime caused intense erythema, cardiac difficulties and symptoms resembling angina pectoris. When chloral was discontinued, the symptoms receded. When 2-4 cg morphine was substituted for chloral in the case of the male patient, the beer was tolerated quite well. However, when treatment with chloral was resumed to effect better sleep, the beer had to be discontinued, as the symptoms recurred. In the case of the female patient, the beer was discontinued only after one of the doctors, upon reading a report of a similar case, connected the attacks of angina pectoris and erythema to intolerance due to interaction.

146. Blöch, J., and Lenhardt, A.
 ADVANTAGES AND DISADVANTAGES IN SHIFTING PATIENTS FROM
 TOLBUTAMIDE TO CHLORPROPAMIDE.
 Ann. N.Y. Acad. Sci. (New York), 74: 954-961 (0 ref.), 1958-59.
 E – general – DC (sensit.) – humans – drug-dep. humans – chronic admin. – in vivo – cardiovasc.
 – skel., muscle, skin – hormones, hormone antag. – *CAAAL-0 A-1263.

Of 200 diabetic inpatients, previously treated with: diet only, tolbutamide and carbutamide, insulin and introductory chlorpropamide, or prolonged insulin, 146 had good control when given chlorpropamide alone. The effect of chlorpropamide in reducing blood and urine sugar was greater than that of the other antidiabetic sulfonylurea preparations, tolbutamide and carbutamide, since lower doses produced equally favourable therapeutic results, as well as a good metabolic equilibrium. Initial daily dosage was 1-1 1/2 g (500 mg doses) and maintenance dosage 0.5 or 0.25 g (often given every other day due to its especially-prolonged hypoglycemic action). Possible success with chlorpropamide was found generally to decrease as the insulin need increased. Of 215 diabetic patients on chlorpropamide (including 15 outpatients), side effects occurred more frequently during early stages of treatment. They consisted of gastro-intestinal disturbances in 10.7% cases, dermatological side reactions in 3.5%, and intolerance to alcohol in 11.6%. 10-15 min after consuming a small quantity of any type of alcohol, flushing, subjectively often felt as "congestion" and a warmth of the face and neck, was visible as a temporary redness sometimes extending to the upper thoracic area in 31 patients. This reaction was similar to an antabuse-alcohol reaction, and could be prevented by avoidance of alcohol.

147. Blomstrand, R., and Theorell, H.
 INHIBITORY EFFECT ON ETHANOL OXIDATION IN MAN AFTER
 ADMINISTRATION OF 4-METHYLPYRAZOLE.

Life Sci. (Oxford), 9(Part 2): 631-640 (20 ref.), 1970.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – dose resp.
 – blood lev. – cardiovasc. – metab. proc. – *CAAAL-0 B-0567.

The effect of 4-methylpyrazole on ethanol metabolism was examined in 5 healthy males (group 1) and in 2 alcoholics (group 2). Group 1 received, on separate occasions, 22.5 ml 47.5% ethanol-1-¹⁴C (5 μ C) po, and ethanol plus 100-1000 mg 4-methylpyrazole po; group 2 received 100 ml 47.5% ethanol-1-¹⁴C (10 μ C) po, and ethanol plus 200 mg 4-methylpyrazole iv. ¹⁴CO₂ excretion was measured, and blood alcohol determinations were made in group 1; in group 2, lactate and pyruvate determinations were made in addition. In group 1 it was found that methylpyrazole inhibited ethanol metabolism, the inhibition reaching a peak 1.5-2 hr after po administration, and decreasing slowly thereafter. 1000 mg methylpyrazole inhibited ¹⁴CO₂ elimination by 49.6% after 1.5 hr, and by 28% after 6 hr. The results indicate a direct relationship between degree of inhibition and dose of methylpyrazole, and an individual variation in response to methylpyrazole, as well. In group 2 a significant decrease in ¹⁴CO₂ was observed during the first 6 hr, followed by prolonged and somewhat increased elimination in the next 6 hr. The blood alcohol concentration was significantly increased, and a more gradual blood alcohol curve produced. The ethanol-induced rise in the lactate-pyruvate ratio was significantly inhibited. Although blood pressure and electrocardiogram observations were made in the experiment, no changes were noted.

148. Blomstrand, R.
 INHIBITORY EFFECT ON ETHANOL OXIDATION IN MAN AFTER
 ADMINISTRATION OF 4-METHYLPYRAZOLE.

Industr. Med. Surg. (Chicago), 39(7): 311 (0 ref.), 1970.
 E – abst. – exp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – dose resp.
 – other drug lev. – metab. proc. – *CAAAL-0 B-0913.

The inhibitory effect of 4-methylpyrazole on ethanol metabolism was investigated in 10 healthy male volunteers. The degree of inhibition was measured by the amount of ¹⁴CO₂ excreted in expired breath after po administration of 5 μ curies ethanol-1-¹⁴C with a carrier dose of 9 g ethanol. Inhibition reached a max 1.5-2 hr after po administration of methylpyrazole, and then decreased slowly. The results suggest an individual response to methylpyrazole, and a dose-response relationship between the degree of inhibition and the dose of methylpyrazole.

149. Blood, F. R.
 ETHYLENE GLYCOL TOXICITY.

Food Cosmet. Toxic. (Oxford), 1: 337-338 (1 ref.), 1963.
 E – exp. – general – DC (unchanged) – mammals – acute admin. – in vivo – other drug lev. – species or sex diff. – metab. proc. – indust. intox. – *CAAAL-10911-D2 A-0557.

One-half of the LD₅₀ of radioactive ethylene glycol (EG) was administered to rats ip. About one-third of the labeled EG was excreted as CO₂ within 48 hr. Simultaneous administration of ethanol, 10 g/kg po or 5 g/kg ip, did not affect the oxidation of EG.

150. Blum, K., Ryback, R. S., and Geller, I.
 EFFECTS OF VODKA AND BOURBON ON SLEEPING TIME IN MICE.

Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 62-66 (15 ref.), 1970.
 E – exp. comp. – congen. stud. – mammals – acute admin. – in vivo – CNS – *CAAAL-12889 B-0502.

0.014 ml of vodka or bourbon/kg body weight, containing approximately equal ethanol concentrations, but with about 70 times as many congeners in the latter beverages, were injected ip into 2 groups

of mice. The mice were then hung from a wire mesh, and fall time, onset time (time to lose righting reflex), and sleeping time were measured. The average results following vodka and bourbon administration, respectively, were as follows: fall time, 86 and 52 sec; onset time, 196 and 64 sec; and sleeping time, 1640 and 2717 sec. 9 vodka-treated mice did not sleep, while all bourbon-treated mice did. In an effort to determine if the presence of the brain neurotransmitter, serotonin, is related to the degree of alcohol effects, p-chlorophenylalanine methyl ester hydrochloride (p-CPA), a serotonin depletor, was given prior to bourbon, vodka, or saline. Insignificant differences in fall, onset, and sleeping times, as compared to alcohol alone, appeared to negate any central role of serotonin. In a third experiment, blood alcohol concentrations in rats injected with bourbon or vodka were found to be similar— 546 ± 47 mg/100 ml after bourbon, and 515 ± 88 mg/100 ml after vodka.

151. Blum, K., Wallace, J., Ryback, R. S., and Geller, I.
AUGMENTATION OF ETHANOL'S EFFECTS IN LABORATORY RATS BY
PRETREATMENT WITH PYRAZOLE.
Pharmacologist (Washington), 12(2): 276 (0 ref.), 1970.
E – abst. – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – mot.
perform. – CNS – metab. proc. – *CAAAL-0 B-0914.

The interaction of ethanol and pyrazole were studied in 3 laboratory test procedures: sleeping time in mice, rotor-rod balance of rats, and lever-pressing behaviour of rats. Pyrazole pretreatment resulted in an increase of ethanol-induced sleeping time. In rotor-rod tests, pyrazole caused an impairment of performance, when administered following an ineffective dose of ethanol. Similarly, in lever-pressing behaviour, an ineffective dose of ethanol caused a marked decline in the performance of pyrazole-pretreated animals.

152. Blumenthal, M., Takki, S. H., and Virolainen, E. S.
CHLORPROTHIXEN INTRAMUSKULÄRT VID SJUKHESBEHANDLING AV
ALKOHOLISTER. [Intramuscular chlorprothixene in hospital treatment of alcoholics].
Nord. Psykiat. T. (Middelfart), 19: 71-73 (7 ref.), 1965.
Da – general – DC (unchanged) – drug-dep. humans – blood lev. – cardiovasc. – tranquilizers –
*CAAAL-11578-N20 B-0252.

Over a period of 6 months, a total of 164 intoxicated alcoholics were treated with im injections of chlorprothixene. The pulse and the blood alcohol concentration were recorded in 20 patients before, and 2 hr after, injection. Only 3 patients developed side effects. Of the 144 other patients, 13 developed nausea and vertigo. All others slept well after the injection of chlorprothixene. The drug, in doses of 30 or 50 mg, is considered safe in the treatment of acutely-intoxicated alcoholics.

153. Bobier, P. -M.
UNE NOUVELLE MÉTHODE RAPIDE DE DÉSINTOXICATION (MORPHINE,
HÉROÏNE, COCAINE, ALCOOL). [A new rapid method of disintoxication (morphine, heroin,
cocaine, alcohol)].
Journal de Médecine de Paris (Paris), 23(3): 47-48 (0 ref.), 1911.
F – general – case hist. – DC (antidotal) – drug-dep. humans – autonomic agents – elect., water-bal.
agents – *CAAAL-0 A-0558.

A successful treatment of 11 cases of alcoholism is reported. The method of detoxification was the following: washing with soap, purgative in conjunction with calomel, then administration of tincture of belladonna, hyoscyamine, and xanthoxylum, in a dose corresponding to the physical condition of the patient and degree of intoxication. Treatment is carried out for 40 hr, increasing the dose by 2 drops/6 hr. 14 hr after the initial dose, a vegetable purgative and calomel is again given in the same dose to effect desired results. A large dose of castor oil is given 6 hr after evacuation, followed immediately thereafter by 10 to 25 cg of codeine phosphate given orally or hypodermically.

154. Boeree, B. H.
SOBERING-UP.
Brit. Med. J. (London), 2: 524-525 (1 ref.), 1961.
E – general – DC (antidotal) – humans – stimulants – *CAAAL-9630-N14 A-0559.

A method of rapid “sobering up” is described which is very transient in nature and of limited application. It is useful when the patient is found comatose or nearly so, smelling of alcohol, and it is important to know either who he is, or whether he has been involved in an accident. It consists of injecting two ml of nikethamide iv. The patient will remain sober enough for 5 to 10 min to give an account of himself before going back to sleep.
155. Bogan, J., and Smith, H.
ANALYTICAL INVESTIGATIONS OF BARBITURATE POISONING—DESCRIPTION OF METHODS AND SURVEY OF RESULTS.
J. Forensic Sci. Soc. (London), 7: 37-45 (11 ref.), 1967.
E – stat. surv. – DC (add., infra-add., unspec. incr.) – post-mort. – humans – blood lev. – other drug lev. – barbiturates – sed., hypnot. – *CAAAL-0 B-0253.

A discussion of the cases of barbiturate poisoning dealt with by the Department of Forensic Medicine, Glasgow University, during the last two yr is given. Methods of identification and measurement are described. It was found that 53% of all fatal barbiturate poisoning cases are complicated by the presence of alcohol. A considerable number of these cases are accidental deaths in which survival might have been possible if alcohol had not been present. The mean blood-barbiturate level in fatal poisoning is reduced by nearly 50% when alcohol is present. The concentration of barbiturate in the brain is shown to be roughly equivalent to that of the blood, whereas the concentration in the liver is about twice as high.
156. Bogen, E.
METHANOL POISONING.
Calif. Med. (San Francisco), 65(5): 230-234 (13 ref.), 1946.
E – exp. cont. – general – DC (unchanged) – humans – mammals – acute admin. – in vivo – acid-base, blood pH, elect. – metab. proc. – alcohols – *CAAAL-4587-A4 A-0560.

Clinical observations in 30 cases of methanol poisoning are presented. Methanol remains longer in the body than ethanol, and results in more permanent disability, especially of vision. The amounts of methanol taken varied from “a few drops” to more than 8 oz of pure methanol. Death resulted after ingestion of large amounts, although one patient who took 6 oz methanol recovered. Besides other medication, ethanol by mouth was tried without observed therapeutic effects. Experiments with guinea pigs were undertaken to further investigate the value of ethanol treatment; 26 guinea pigs were given 30 g ethanol ip, 26 were given 30 g methanol ip, and 48 were given combinations of both in amounts totaling the same as that of the single doses in the previous sets. 18 of the ethanol group died in 6 hr; 8 of the methanol group died in 6 hr, and 18 of the ethanol-methanol group died in 6 hr. It is concluded that the greater toxicity of ethanol and its futility in methanol poisoning are evident from the results.
157. Böhm, E.
ERSTMALIGE KLINISCHE UND MORPHOLOGISCH-PATHOLOGISCHE BEOBACHTUNGEN NACH 15 G ISONIKOTINSÄUREHYDRAZID-EINNAHME BEI GLEICHZEITIGER ALKOHOLVERGIFTUNG. [Initial clinical and morphological-pathological observations after 15 g isonicotinic acid hydrazide intake, with simultaneous alcohol poisoning].
Dissertation, Medical Faculty of the University of Jena, East Germany, 31 pp. (54 ref.), 1953.
G – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – humans – cardiovasc. – CNS – liver, kidney – anti-infectants – *CAAAL-0 A-0561.

A study is made of a case history of a 19 yr-old patient who attempted suicide with 15 g isonicotinic acid hydrazide (INH) following intoxication by alcohol. 30 min later the patient manifested an appreciable state of intoxication with ensuing coma. Coronary insufficiency was established via electrocardiogram 15 hr after drug intake (150 tablets). Treatment with narcotics, administered iv, had no effect on the appreciable muscular spasm. Death occurred in deep coma 16 hr after drug intake. It is concluded, on the basis of observations following a lethal INH dose under conditions of alcohol intoxication, that INH in high doses is a hypoxydose cell poison.

158. Böhmer, K.

DIE EINWIRKUNG EINIGER ARZNEIMITTEL AUF DEN VERLAUF DER

BLUTALKOHOLKURVE. [The action of some medicaments on the blood alcohol curve].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 30: 205-217 (10 ref.),

1938.

G – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – acute admin.

– in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – metab. proc. – analg., antipyret.

– anti-infectants – hormones, hormone antag. – *CAAAL-874-A1

A-0562.

The influence of aspirin, pyramidon, quinine, and insulin on the blood alcohol curve in man were studied. The subjects received between 0.993 and 1.115 g of absolute alcohol/kg. Aspirin and pyramidon diminished and retarded the absorption, and delayed the catabolism, of alcohol; quinine had no effect; insulin prevented the normal absorption and delayed the excretion. The test results are discussed in connection with road traffic.

159. Boissier, J. -R., Simon, P., and Le Bourhis, B.

ACTION PSYCHOTROPE DU TRANS ANÉTHOLE ET DE L'ÉTHANOL ADMINISTRÉS CONJOINTEMENT À LA SOURIS. [Psychotropic action of trans-anethole and ethanol, jointly administered to the mouse].

Ann. Nutr. (Paris), 23: 215-222 (7 ref.),

1969.

F – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp.

– CNS – gastrointest. agents – stimulants – *CAAAL-14306

B-1007.

Male mice (18-22 g) received various doses of anethole (in the form of an aqueous emulsion, containing 1% tween 20) and/or an aqueous ethanol sol; sol of both substances were such that the total vol received by each animal was 20 ml/kg. The following tests were conducted: measurement of acute toxicity after po or ip administration, measurement of hypnotic activity after ip administration, and measurement of anticonvulsant activity after po or ip administration—the convulsant agent being electroshock, pentetrazole (an aqueous sol containing 5 mg/ml, perfused iv at 0.005 ml/sec), or flurothyl (0.05 ml of a flurothyl sol diluted to 1:75 in polyethylene glycol 400). In addition, the authors sought to determine whether the combined anethole-ethanol effect was additive, supra-additive, or antagonistic. The results showed that the psycholeptic effects of anethole in mice are not significantly modified by ethanol, whether the po or ip routes are used. The combined effect is generally additive, with non-significant trends towards supra-addition or antagonism, according to dosage and route of administration. It is concluded that, since anisated alcoholic beverages are comprised of 40-45% ethanol plus a maximum of 0.2% anethole, the possible toxicity, hypnotic effects, and anticonvulsive effects resulting in humans from an interaction of these 2 compounds are considered to be a negligible hazard.

160. Bonjour, J. P., Peters, G., Chomety, F., and Regoli, D.

RENAL EFFECTS OF VAL₅-ANGIOTENSIN II-AMIDE, VASOPRESSIN AND DIURETICS IN THE RAT, AS INFLUENCED BY WATER DIURESIS AND BY ETHANOL ANESTHESIA.

Europ. J. Pharmacol. (Amsterdam), 2: 88-105 (57 ref.),

1967.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals –

acute admin. – in vivo – other drug lev. – species or sex diff. – absorp., distrib., stor. – cardiovasc. – liver, kidney – metab. proc. – autonomic agents – elect., water-bal. agents – hormones, hormone antag. – *CAAAL-0 B-0254.

The conditions which favour one or the other of the three types of renal effects of val₅-angiotensin-II-amide in the rat, the dog, or in man are discussed. Ethanol anesthesia profoundly modified the renal effects of angiotensin in water-loaded rats. During the first 10 to 20 min of infusion of angiotensin II-amide, the glomerular filtration rate (GFR), sodium excretion, and CH₂O fell. With continued infusion, GFR reverted to normal, while sodium excretion increased less than in unanesthetized animals; simultaneously, a vasopressin-like depression of CH₂O appeared. Ethanol anesthesia enhanced the anti-diuretic effect of angiotensin and increased the sensitivity of water-loaded rats to the anti-diuretic action of vasopressin by about 1 order of magnitude, but did not influence the diuretic response to hydrochlorothiazide or furosemide.

161. Bonnichsen, R., Dimberg, R., Maehly, A., and Åqvist, S.
 DIE ALKOHOLVERBRENNUNG BEI ALKOHOLIKERN UND BEI ÜBRIGEN
 VERSUCHSPERSONEN: METHODIK, ERGEBNISSE UND KURZE THEORETISCHE
 DISKUSSION. [Alcohol metabolism in alcoholics and various test subjects: method, results, and
 short theoretical discussion].
 Blutalkohol (Hamburg), 5(5): 301-317 (7 ref.), 1968.
 G – exp. comp. – DC (unchanged) – humans – drug-dep. humans – acute admin. – in vivo – blood
 lev. – absorp., distrib., stor. – liver, kidney – metab. proc. – sed., hypnot. – tranquilizers – *CAAAL-0
 B-0915.

The rate of alcohol metabolism (R) was investigated in adult alcoholic patients (29-58 yr), juvenile habitual drinkers (17-30 yr), and normal, healthy humans. Alcohol, in the form of beer or liquor (40% alcohol), was ingested po on an empty stomach. R was determined enzymatically and by gas chromatography in whole blood and plasma. In phase 1 of the experiments, the effects of the following drugs on R were determined in alcoholics: librium (10 or 50 mg), diminal duplex (0.2 g vinbarbital plus 0.1 g aprobarbital), chlorprothixene (50 mg), chlomethiazole (1.5 g), or methaqualone (0.5 g). It was found that R was unaffected by the drugs tested. The values of R for alcoholics were significantly higher (14-16 g/hr) than were those of moderate drinkers (8-11 g/hr), and liver disorders were indicated in the former. Juvenile habitual drinkers showed high blood alcohol concentrations (BAC), but the R values were relatively low (5-9 g/hr), and no significant liver damage was found. It is concluded that, compared to moderate drinkers, alcoholics can tolerate a high BAC, and have a high R. Both of these phenomena are correlated with an increased tolerance of alcoholics to alcohol.

162. Boothby, W. M.
 ETHER PERCENTAGES.
 J.A.M.A. (Chicago), 61(11): 830-834 (2 ref.), 1913.
 E – SEC – cross-tol. – drug-dep. humans – respir. – anesthetics – *CAAAL-0 A-0563.

A description of a technique for determining the percentage of ether in air during the administration of an anesthetic is given. It is noted that the well-known increase in depth of respirations in alcoholics appears to be due to the greater excitatory power of ether on the respiratory center in such subjects. Clinically, it is known that large, vigorous, alcoholic patients require a great quantity of ether poured on the mask to produce anesthesia. Physiologically, it is recognized that the same tension of ether vapour in the body produces in all subjects the same depth of anesthesia. The apparent discrepancy between clinical experience and the physiologic law is said to consist in the difficulty of the anesthetist in bringing the large volume of air respired by deep-breathers up to the percentage of ether required.

163. Borden, T. A., and Bidwell, C. D.
TREATMENT OF ACUTE ETHYLENE GLYCOL POISONING IN RATS.
Invest. Urol. (Baltimore), 6(2): 205-210 (33 ref.), 1968.
E – exp. cont. – exp. comp. – DC (antidotal) – post-mort. – humans – mammals – acute admin. –
in vivo – species or sex diff. – acid-base, blood pH, elect. – liver, kidney – metab. proc. – gastrointest.
agents – *CAAAL-13372 B-0503.

Treatment of ethylene glycol (EG) poisoning with sodium bicarbonate (NaHCO_3) and ethanol is discussed. Liver-alcohol dehydrogenase degrades ethylene glycol to oxalate, some of which is subsequently deposited in the kidney as calcium oxalate. Theoretically, ethanol competes with EG for enzymatic degradation, and thereby decreases its conversion to toxic oxalate. NaHCO_3 helps correct the frequent acidosis associated with EG poisoning, as well as to decrease calcium excretion. Effective calcium concentration is further reduced by increased citrate excretion, which complexes with calcium and is enhanced in alkaline environments. Groups of male rats, poisoned with pure EG (10 ml/kg body weight-minimum lethal dose), either received no treatment, or, 15 min later and at 6-6 hr intervals, ip injections of 0.2 g NaHCO_3 or 0.4 g ethanol, separately or in combination. The following rats survived at least 96 hr: 14% (10 of 71) receiving no treatment; 71% (22 of 31) receiving only NaHCO_3 ; 73% (25 of 34) receiving only ethanol; and 89% (25 of 28) receiving the combination. Diuresis was not a factor. EG (5 ml/kg) caused calcium oxalate crystals in renal parenchyma of most rats receiving no treatment, NaHCO_3 caused less crystal formation than ethanol, and the combination none. Humans probably metabolize EG similarly, and ethanol- NaHCO_3 treatment is suggested for acute EG poisoning.

164. Bouchier, I. A. D., and Williams, H. S.
DETERMINATION OF FAECAL BLOOD-LOSS AFTER COMBINED ALCOHOL AND
SODIUM ACETYLSALICYLATE INTAKE.
Lancet (London), 1(7587): 178-180 (21 ref.), 1969.
E – exp. cont. – DC (unchanged) – humans – other org. – in vivo – dose resp. – other drug lev. –
absorp., distrib., stor. – acid-base, blood pH, elect. – G.I. tract – analg., antipyret. – *CAAAL-13346
B-0495.

22 healthy adult males were given a buffered solution of sodium acetylsalicylate (Alka-Seltzer) and alcohol, separately and together, in a double-blind controlled study to determine if an increase in the normal faecal blood-loss resulted. It was found that the acetylsalicylate preparation provided sufficient buffering capacity to maintain the gastric contents at a pH level at which no gastric absorption of acetylsalicylate occurs, and, therefore, no mucosal damage with bleeding takes place. After labelling each subject's erythrocytes with radioactive chromium, daily stool collections were taken for 16 days. The normal individual blood loss was determined over the first 4 days. On the evening of the fourth day, each subject prior to his meal took an equivalent of 142 ml of 40% alcohol, followed 5 min later by 728 mg sodium acetylsalicylate or a placebo in 200 ml of water. On the tenth day, the acetylsalicylate and placebo administrations were reversed after consumption of the same amount of alcohol as before. Mean daily faecal blood-losses were: 0.36 ± 0.05 ml for the 4 day control period, 0.40 ± 0.09 ml after taking alcohol and placebo, 0.45 ± 0.05 ml after taking alcohol and sodium acetylsalicylate. Upon analysing the results of the 3 groups, no significant statistical differences were found.

165. Bourne, H. B., Ross, R. C., and Philpott, N. W.
INTRAVENOUS ALCOHOL IN OBSTETRICAL LABOUR.
Canad. Med. Ass. J. (Toronto), 69: 250-253 (3 ref.), 1953.
E – general – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – CNS – skel.,
muscle, skin – analg., antipyret. – elect., water-bal. agents – *CAAAL-6754-V10 A-0564.

A series of 100 primiparae is presented in whom 5% (250 to 300 cc) and 7 1/2% (150 cc) alcohol sol mixed with glucose and protein hydrolysate was used as an analgesic agent, either alone or in

conjunction with 100 mg demerol im; iv alcohol, either alone or with small doses of demerol, gave better results than demerol alone. The best results were obtained when 5% alcohol and 50 mg demerol were used simultaneously. Intravenous alcohol seems to be a safe and potent analgesic agent in obstetrics, and has no apparent adverse effect upon the fetus.

166. Bourrinet, M. P.

INFLUENCE DE L'ALCOOLISATION CHRONIQUE SUR L'ACTIVITÉ DE LA CHLORPROMAZINE CHEZ LA SOURIS. [The effect of chronic alcoholization on chlorpromazine activity in the mouse].

Ann. Pharm. Franc. (Paris), 27(7-8): 543 (0 ref.),

1969.

F – abst. – cross-tol. – mammals – mot. perform. – metab. proc. – tranquilizers – *CAAAL-0

B-0568.

In experiments on mice fed an alcohol diet for more than 8 months, it was found that the depressive effect of chlorpromazine was increased and prolonged. It is suggested that chronic alcohol intake for this length of time produces a decreased hematic metabolism of chlorpromazine.

167. Bourrinet, P.

ÉTUDE EXPÉRIMENTALE DE L'INFLUENCE DE L'ALCOOLISATION AIGUE ET CHRONIQUE SUR L'ACTIVITÉ DES MÉDICAMENTS. [Experimental study of the influence of acute and chronic alcohol administration on the activity of drugs].

Revue de l'Alcoolisme (Paris), 10: 186-198 (20 ref.),

1964.

F – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin.

– chronic admin. – in vivo – absorp., distrib., stor. – cardiovasc. – CNS – metab. proc. – nerv. syst.

– analg., antipyret. – anesthetics – barbiturates – stimulants – tranquilizers – *CAAAL-0

A-1375.

Acute intoxication was produced in rabbits, guinea pigs, rats, and mice by administration of 1 ml/100 g 40% ethanol sol po, and, 30-60 min later, a test drug was administered. Chronic intoxication was induced in rabbits and guinea pigs by daily po administration (5 days/week for 3 months) of 0.4 ml/100 g 40% ethanol sol in rabbits and 0.5 ml/100 g 40% ethanol in guinea pigs. The 4 categories of drug action investigated were drug effects on: the CNS, the autonomic nervous system, histamine and antihistamines, and the cardiovascular system. The first category consisted of: general anesthetics and hypnotics (ether, urethane, pentobarbital, and penthiobarbital), local anesthetics (procaine hydrochloride and cocaine hydrochloride), analgesics (morphine hydrochloride, pethidine hydrochloride, and pyrrolamidol), CNS stimulants (nikethamide, lobeline, ephedrine, and aminophylline), neuroleptics and tranquilizers (chlorpromazine, reserpine, butyrophenone, meprobamate, and methylpentynol carbamate), and convulsant agents and antiepileptics (electroshock, pentetrazole, and phenobarbital). In the second category were adrenalin, noradrenalin, ephedrine, ergotamine, yohimbine, acetylcholine, and atropine. The third category included histamine, mepyramine, and promethazine, and in the fourth category were digitalin and horse-chestnut extract. It is concluded that acute and chronic alcohol intoxication are absolutely distinct pathological states with different consequences. Acute alcohol intoxication augments the action of general anesthetics and hypnotics, local anesthetics, analgesics, neuroleptics and tranquilizers, and antiepileptics. Chronic intoxication enhances the effect of general and local anesthetics, increases sensitivity to experimental epilepsy, decreases antiepileptic action, increases sensitivity to acetylcholine and histaminic bronchospasm, augments digitalin toxicity, and increases the capillary permeability, but decreases the vasotonic activity, of horse-chestnut extract.

168. Bourrinet, P.

INFLUENCE DE L'ALCOOLISATION CHRONIQUE SUR L'ACTIVITÉ DE LA CHLORPROMAZINE CHEZ LA SOURIS. [Influence of chronic alcoholization on

chlorpromazine effect in mice].

Ann. Pharm. Franc. (Paris), 27(12): 729-732 (12 ref.),

1969.

F – exp. cont. – cross-tol. – acute admin. – chronic admin. – mot. perform. – liver, kidney – metab. proc. – tranquilizers – *CAAAL-0 B-0504.

Behavioral changes in motility, activity, and equilibrium caused by chlorpromazine were tested in alcoholic and non-alcoholic mice. All alcoholic mice used were alcoholized over a period of 7 to 8 months. Before testing, mice were grouped according to chlorpromazine dosage injected (0.5, 1, 2, 3, or 5 mg/kg sc). Motility was tested by allowing the mice free movement in a box, and then counting the number of times that vertical beams of light were broken. Motility was tested up to 4 hr after injection. Equilibrium was tested by suspending mice by their hind legs, and, having lowered the mice, then timing the re-establishment of the normal standing position. Muscle tone was tested by placing the mice on a rotating rod and timing their ability to avoid falling. Tests showed that the activity-depressing effect of chlorpromazine was increased in alcoholized mice. Motility was not modified, but clear evidence of decreased muscle tone and loss of equilibrium was found. The depressive action lasted longer in alcoholic mice, suggesting that the livers of these animals took longer to metabolize the chlorpromazine. It is concluded that this abnormal retention must be considered when phenothiazine derivatives are prescribed for alcoholics.

169. Boutigny, P. -H.

SUR L'ACTION QU'EXERCE L'AMMONIAQUE EMPLOYÉE CONTRE L'IVRESSE. [On the effect of ammonia administered for intoxication].

Journal de Chimie Médicale, de Pharmacie et de Toxicologie (Paris), 10: 532-535 (0 ref.), 1834.

F – general – DC (antidotal) – humans – acid-base, blood pH, elect. – metab. proc. – miscellaneous – *CAAAL-0 A-0565.

Case material is given concerning an alcohol-intoxicated man treated with ammonium carbonate to neutralize the alcohol. The author points out his awareness of existing contradictions regarding the effectiveness of this remedy, and poses a number of physico-chemical considerations—e.g., whether the nerves are good chemical conductors; whether alcohol, carbonic acid, and some hydrocarbons, which form during the fermentation of sugared liquors, are electronegative, attracting in the digestive system positive electricity, and repelling negative electricity in the brain; and whether this electric influence causes perturbations which cease when neutralization takes place. The author suggests this explanation of the neutralization of volatile substances (e.g. alcohol, ammonia).

170. Bowes, H. A.

THE ROLE OF LIBRIUM IN AN OUT-PATIENT PSYCHIATRIC SETTING.

Dis. Nerv. Syst. (Galveston), 21(3) Suppl.: 20-22 (0 ref.),

1960.

E – SEC – general – DC (unchanged) – psychot. humans – tranquilizers – *CAAAL-0 A-1265.

The role of librium in treating disturbed patients in an out-patient setting is discussed. The author states his own conviction that more than 90% of patients presently being admitted to mental hospitals could have been treated on this basis. Librium, while specifically indicated in acute situational anxiety tension states, is also effective in long-standing anxiety and phobic reactions, and even in some obsessional states. Librium is a remarkably safe drug which does not appear to potentiate alcohol. 1 disturbed patient took 300 mg of librium plus a pint of whiskey during an evening, and, though confused and ataxic the next day, was back to normal within 36 hr.

171. Boyd, E. M.

A SEARCH FOR DRUGS WITH DISULFIRAMLIKE ACTIVITY.

Quart. J. Stud. Alcohol (New Haven), 21: 23-25 (3 ref.),

1960.

E – exp. cont. – DC (unchanged) – DC (sensit.) – humans – acute admin. – in vivo – cardiovasc. – unclass. ther. agents – *CAAAL-8630-B1 A-1376.

71 drugs were screened for the ability to produce disulfiram-like actions in humans after alcohol intake. Each drug was administered po to volunteers for 4 days. On the fourth day, alcohol (90 ml of a fortified port wine, containing 18.2% alcohol by vol) was given po 2 hr after administration of the last drug dose. The number of volunteers in each drug group ranged from 4 to 25. Disulfiram-like reactions were recorded after amodiaquine hydrochloride, ephedrine hydrochloride, phenazone, phentolamine hydrochloride, and tolazoline hydrochloride. 1 person in each of the groups receiving amodiaquine, ephedrine, and phenazone experienced nausea 1/2 to 2 hr after alcohol. 1 person receiving phentolamine had a flushed skin 15 min after alcohol. Tolazoline at 1/4 to 1 1/2 hr after alcohol, produced a tingling in the head region in 6 of 7 subjects, and a feeling of warmth and fullness in the head in 4 of the 7 subjects. The incidence of reactions after alcohol was significant only in the tolazoline group.

172. Branch, A., and Tonning, D. J.
ACUTE METHYL ALCOHOL POISONING: OBSERVATIONS IN SOME THIRTY CASES.
 Canad. J. Public Health (Toronto), 36: 147-151 (5 ref.), 1945.
 E – SEC – general – DC (antidotal) – post-mort. – humans – blood lev. – absorp., distrib., stor. – acid-base, blood pH, elect. – alcohols – *CAAAL-4148-C4 A-0566.

A treatment of 30 cases of methanol poisoning is described. The average methanol blood content at admission was 0.098%. Symptoms, treatment, and physical and laboratory findings are presented in detail. Some of the patients received ethyl alcohol (no dosage given) in accordance with the theory advanced by Røe (Acta Med. Scand. (Stockholm), 113(6): 558-608, 1943) that ethyl alcohol is absorbed more readily by cells than is methyl alcohol, and that, consequently, the latter, being absorbed slowly, is eliminated without causing toxic effects; however, no conclusions could be drawn. Several of those who recovered were known to have drunk ethyl alcohol in one form or another after drinking the methyl alcohol.

173. Brandino, G.
RICERCHE SPERIMENTALI SULL'AZIONE RECIPROCA FRA ALCOOL ETILICO E STRICNINA. [Experimental investigation of the interaction of alcohol and strychnine].
 Archivio di Anthropologia Criminale, Psichiatria, Medicina Legale e Scienze Affini (Turin), 57(Suppl): 288-289 (0 ref.), 1937.
 I – exp. comp. – DC (decrease) – DC (unchanged) – mammals – other org. – acute admin. – in vivo – CNS – stimulants – *CAAAL-0 A-0567.

Experimental investigations were carried out on the interaction of ethanol and strychnine in a series of tests with frogs, rabbits, and dogs, to which alcohol was administered ip, hypodermically, and iv prior to strychnine (in the first series of tests). In the second series of tests, alcohol and strychnine were introduced simultaneously. In the third series, alcohol was administered after strychnine. The following conclusions were derived: ethanol administered prior to strychnine counteracts the onset of convulsions—the animals survived greater-than-lethal doses of strychnine; strychnine administered in lethal doses prior to alcohol was fatal to animals, but they survived simultaneous administration of ethanol and strychnine; acute ethanol intoxication (lethal dose) was not influenced by strychnine (the animals remained asleep and finally died).

174. Braun, H.
UNTERSUCHUNGEN ÜBER EIN "ENTRAUSCHUNGSMITTEL". [Examination of a "detoxifying drug"].
 Arch. Pharm. (Weinheim), 285: 349-352 (0 ref.), 1952.
 G – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – mot. perform. – indust. intox. – *CAAAL-6383-A1 A-0568.

Bavarin 404, an alleged anti-intoxicant, was administered to 4 healthy human subjects (2 men, 2 women) on 2 occasions (8-14 days apart), after ingestion of 38% brandy (1 cc ethanol/kg). On the second occasion, bavarin 404 was given 1 hr after alcohol. A typing performance test was then given, and blood samples taken 1, 2, and 3 hr after alcohol; alcohol determinations were made according to the Widmark method. Bavarin 404 failed to affect the duration of intoxication or the blood alcohol level. On both occasions the blood alcohol curves for each subject were nearly identical.

175. Briglia, R. J.

TOXICOLOGICAL SCREENING PROGRAM OF CORONER'S CASES IN SACRAMENTO COUNTY.

Sacramento County Coroner's Office, Sacramento, California, U.S.A., 14 pp. (3 ref.), 1966.
E – stat. surv. – DC (add, infra-add., unspec. incr.) – med.-leg. – post-mort. – humans – blood lev.
– analg., antipyret. – barbiturates – sed., hypnot. – *CAAAL-0 B-0255.

Of a total of 1,618 coroner's cases, all were analyzed for alcohol (gas chromatography), and 1,157 cases for barbiturates (ultra-violet spectrophotometry). 23.6% of the cases showed positive alcohol levels, and 9.3% showed positive barbiturate levels. In a number of cases in which drugs directly contributed to the cause of death, alcohol and barbiturates were found in combination. Other drug combinations (with alcohol) causing death in a few cases included paraldehyde, morphine, and carbon monoxide.

176. Brodie, B. B., Shore, P. A., and Silver, S. L.

POTENTIATING ACTION OF CHLORPROMAZINE AND RESERPINE.

Nature (London), 175(4469): 1133-1134 (4 ref.), 1955.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – other drug lev. – absorp., distrib., stor. – CNS – metab. proc. – tranquilizers – *CAAAL-8842-D2 A-0569.

Reserpine (5 mg/kg) and chlorpromazine (5 mg/kg) were shown to potentiate the hypnotic action of hexobarbitone (100 mg/kg) and ethanol (5 g/kg in 50% sol) administered to mice ip. Neither potentiating agent influenced the rate of metabolic transformation of the hypnotics, but each presumably acted by increasing the sensitivity of the central nervous system.

177. Brodie, B. B., and Shore, P. A.

A CONCEPT FOR A ROLE OF SEROTONIN AND NOREPINEPHRINE AS CHEMICAL MEDIATORS IN THE BRAIN.

New York Academy of Sciences, Annals (New York), 66: 631-642 (20 ref.), 1957.
E – SEC – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – CNS – metab. proc. – hallucinogens – tranquilizers – *CAAAL-0 A-1377.

The concept is developed that a number of mutually antagonistic centers that regulate homeostatic mechanisms in the brain are innervated by serotonergic and adrenergic nerve fibres. Although reserpine and chlorpromazine can produce similar central effects, they do so by different mechanisms. Certain differences in action of these 2 substances are noted, including a difference in the nature of their potentiating action on certain hypnotics. In 1 experiment, adult male mice were divided into 5 groups and given various drug combinations ip. Group 1 (control) received 4 g/kg ethanol. Groups 2 and 3 were given 5 mg/kg reserpine 1 hr before the ethanol dose, group 3 receiving in addition 10 mg/kg LSD in 2 divided doses 1 hr before and simultaneously with ethanol. Groups 4 and 5 were given 5 mg/kg chlorpromazine simultaneously with ethanol, group 5 also receiving 10 mg/kg LSD in 2 divided doses 1 hr before and simultaneously with the administered combination. LSD by itself had virtually no effect on the duration of hypnosis produced by ethanol. The times from loss to return of righting reflex in groups 1, 2, 3, 4, and 5 were, respectively: 40 min, more than 300 min, 54 min,

150 min, and 147 min. Thus both reserpine and chlorpromazine were found to have marked activity, but only that of reserpine was blocked by LSD.

178. Brohult, J., Lennart, L., and Reichard, H.
 URINARY EXCRETION OF ADRENAL HORMONES IN MAN: EFFECTS OF
 ETHANOL INGESTION, AND THEIR MODIFICATION BY CHLORMETHIAZOLE.
 Acta Med. Scand. (Stockholm), 188(1-2): 5-13 (49 ref.), 1970.
 E – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – glands – liver, kidney – metab.
 proc. – hormones, hormone antag. – tranquilizers – *CAAAL-0 B-0916.

As chlormethiazole (heminevrin) is effective in treatment of withdrawal symptoms in alcoholics, psychomotor agitation, and anxiety states, its effect on adrenal function after alcohol intake was studied. About 500 ml whisky (3 g ethanol/kg) was given to 9 males; in addition, heminevrin (0.5 g at bedtime, plus 1 g on the next evening) was given to 5, and placebos to 4. Ethanol induced increased adrenaline excretion, but, 12 hr later, the non-treated group excreted significantly more adrenaline than did the treated. Heminevrin also modified the alcohol-induced rise in noradrenaline excretion in the hangover period. No significant increase in 17-hydroxycorticosteroids occurred, except on days 2, 4, and 6 in the heminevrin group. There was a 53% decrease in 17-ketosteroid excretion in the post-ethanol period in both groups. The peak for adrenaline excretion was in the ethanol period, and, for noradrenaline, in the hangover period. The results indicate prolonged adrenal cortical and medullary stimulation, and show a modifying effect of heminevrin on sympathomedullary, but not necessarily adrenocortical, reactions after ethanol ingestion. Alternative hypotheses to implied ethanol-induced direct or indirect stimulation of the adrenal medulla and sympathetic nervous system are discussed.

179. Broser, F.
 ÜBER DIE ÄTHERSUCHT UND IHRE BEZIEHUNGEN ZUM CHRONISCHEN
 ALKOHOLISMUS. [Ether addiction and its relationship to chronic alcoholism].
 Nervenarzt (Berlin), 20(3): 113-122 (50 ref.), 1949.
 G – review – conj. addict. – DC (add., infra-add., unspec. incr.) – drug-dep. humans – anesthetics
 – *CAAAL-5184-L3 A-0570.

Case material previously reported is reviewed with respect to the casuistics of ether addiction and its manifestations in relation to other addictive agents. Synergism between alcohol and ether occurred when alcohol was ingested following inhalation of ether. Contrary to earlier conclusions, the author feels that ether addiction bears no relation to alcoholism. He explains that earlier writers did not recognize that, not ether addicts, but chronic alcoholics were involved in the cases cited, alcoholics who either used ether as a substitute because of the lower price, or mixed it with alcohol.

180. Brown, D. J., Hughes, F. W., Forney, R. B., and Richards, A. B.
 EFFECT OF D-AMPHETAMINE AND ALCOHOL ON ATTENTIVE MOTOR
 PERFORMANCE IN HUMAN SUBJECTS.
 In: Harger, Rolla N., ed. *Alcohol and Traffic Safety*. Proceedings of the Fourth International
 Conference on Alcohol and Traffic Safety at Indiana University, December 6-10, 1965. Bloomington,
 Indiana: Indiana University Press, pp. 215-219 (0 ref.), 1966.
 E – exp. cont. – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – mot. perform.
 – CNS – amphetamines – *CAAAL-0 B-0256.

In double-blind experiments on 40 human subjects, attentive motor performance was measured while subjects experienced the depression of alcohol (45 ml/150 lb), the stimulation of amphetamine (5 mg), and the effect of both drugs combined. During stress d-amphetamine failed to affect the alcohol-depressed subjects, and did not improve driving skills.

181. Brown, E. A., Hunter, J. M., and Maling, H. M.
SOME EFFECTS OF ETHANOL ON THE DISPOSITION OF PALMITATE BY INTACT
AND ADRENALECTOMIZED RATS.
Proc. Soc. Exp. Biol. Med. (New York), 122: 1079-1085 (29 ref.), 1966.
E – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
– in vivo – blood lev. – blood comp., sites, lymph – liver, kidney – *CAAAL-12884 B-0505.

It was observed that the administration of a single dose of ethanol or other agents, such as carbon tetrachloride or ethionine, produces a triglyceride fatty liver in intact, but not in adrenalectomized (ADX) rats. The purpose of the experiments was to compare in intact and ADX rats the incorporation of labelled palmitic acid into liver lipids, its excretion as $C^{14}O_2$, and the effect of ethanol on these processes. Female rats weighing about 160-180 g were used. Ethanol (4 or 6 g/kg) was given po in a 50% aqueous sol; 2 or 4-5 hr after ethanol, 0.25 ml palmitate-1- C^{14} was injected into the tail vein. Blood ethanol levels were determined. 1 hr after the palmitate injection, the liver was removed and the hepatic lipid content determined. In ADX rats, a depressed general metabolism was observed at high blood ethanol levels. The results are tabulated and graphically evaluated. The authors conclude that ADX rats, as well as intact rats, oxidize the free fatty acids and incorporate them into liver lipids, and that ethanol increases the palmitate-1- C^{14} in the liver lipids 1 hr after administration, but decreases the amount oxidized to CO_2 in both groups of animals.

182. Bruns, O.
I. PERVITIN: PHARMAKOLOGIE UND KLINIK. II. PERVITIN: DIE FRAGE NACH
DER LEISTUNGSSTEIGERUNG. [I. Pervitin: Pharmacological and clinical aspects. II.
Pervitin: the question of performance enhancement].
Fortschritte der Therapie (Berlin), 17: 37-44 and 90-100 (33 ref.), 1941.
G – review – DC (decrease) – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol.
perform. – amphetamines – *CAAAL-0 A-0571.

The literature on the pharmacology of pervitin and its clinical and non-clinical use, as reflected in practice and research, is reviewed. The prophylactic value of pervitin against alcohol is mentioned; the drug is supposed to increase tolerance toward alcohol and have a good sobering effect. Blood alcohol concentrations were not influenced by pervitin, and yet, psycho-technical tests showed almost normal sobriety performances despite the alcohol. A number of experiments are reviewed which were carried out to answer the question whether pervitin is capable of improving human performance (mental as well as physical) subjectively or objectively.

183. Brunton, L., and Tunnicliffe, F. W.
CONCERNING CERTAIN APPARENTLY INJURIOUS CONSTITUENTS OF POTABLE
SPIRITS.
Lancet (London), 2: 1643-1644 (0 ref.), 1900.
E – exp. – congen. stud. – humans – mammals – acute admin. – in vivo – other drug lev. – species
or sex diff. – CNS – respir. – skel., muscle, skin – *CAAAL-0 A-1265.

The physiological action of furfural was studied in cats, dogs, rabbits, and 2 men. A transient cycle of symptoms in animals given 0.1-0.05 g sc resulted: paralysis of voluntary muscles, followed by clonic and tonic convulsions (probably due to asphyxia), and rapid, irregular breathing. Smaller doses caused transient irritation (e.g., ataxia, tremors and twitching), while larger doses caused respiratory muscle paralysis, asphyxia and death. Recovery from a profoundly intoxicated state in animals was more sudden and accompanied by fewer secondary symptoms (restlessness, discomfort, disinterest in food, bad temper) after the administration of aldehyde-free spirit. Until sober, they slept, after which they appeared perfectly normal. Thus, disagreeable symptoms analogous to those in humans appear more due to aldehydes in the spirit than to the spirit itself or to higher alcohols. 2 men given 0.1 g furfural

experienced a pain at the back of the neck extending to the occipital region, and a throbbing and pulsation in the head, although pulse and respiration were normal.

184. Brusch, C. A., Cerrato, C. M., Papas, P. N., and Straccia, F. A.
 CLINICAL AND LABORATORY EVALUATION OF ALCOHOLIC BEVERAGES.
 Amer. J. Proctol. (New York), 6(2): 140-144 (4 ref.), 1955.
 E – exp. cont. – exp. comp. – congen. stud. – humans – mammals – acute admin. – in vivo – dose
 resp. – blood lev. – psychol. perform. – CNS – G.I. tract – respir. – *CAAAL-7367-V1 A-0572.

Each of 20 human subjects drank one of: vodka, gin, rum, or bourbon on 4 different occasions. All drinks contained 3 oz of absolute alcohol, diluted to 12 oz with water. Although the blood alcohol levels were comparable after all drinks, the degree of intoxication was slightly lower after vodka. Hangovers occurred only twice after vodka, compared to 16, 17, and 19 times after the other beverages. In addition, mixtures of congeners corresponding to the amounts found in whiskey, vodka, gin, or rum were prepared and tested on mice, rats, and rabbits.

185. Brzeski, Z., and Kuczyński, L.
 ZATRUCIE ŚMIERTELNĄ DAWKĄ INH W UPOJENIU ALKOHOLOWYM [Poisoning
 with a lethal dose of INH during alcohol intoxication].
 Wiad. Lek. (Warsaw), 21: 405-407 (18 ref.), 1968.
 Po – ES – RS – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – respir. – metab.
 proc. – anti-infectants – barbiturates – elect., water-bal. agents – musculoskel. agents – nutritive agents
 – tranquilizers – *CAAAL-0 B-0257.

Case material is presented concerning an attempted suicide by ingestion of 240 mg/kg (300 tablets) isonicotinic acid hydrazide (INH), following intoxication with alcohol. The patient was subjected to hospital treatment, while unconscious, with exchange transfusion, controlled and assisted respiration, oxygen therapy and drugs (phenactyl, hydrocortisone, luminal, vitamins, antibiotics, and 40% glucose sol). The patient was released from the hospital after 6 days. It is concluded that the toxicity of INH was increased in the presence of alcohol, inhibiting its oxidation to the acetaldehyde stage. The recovery of the patient is attributed to exchange transfusion and full oxygen therapy following intubation and assisted respiration.

186. Bunn, L.
 ÜBER DEN EINFLUSS VON "ELEKTROVID[SIC] UND POLYSAN" AUF DEN
 BLUTALKOHOLSPIEGEL UND DIE ALKOHOL-BEDINGTE PSYCHOMOTORISCHE
 LEISTUNGSMINDERUNG. [The influence of "elektrovit" and "polysan" on the blood alcohol
 level, and on the decrease of psychomotor performance caused by alcohol.]
 Dissertation, Medical Faculty of the University of Heidelberg, West Germany, 51 pp. (53 ref.), 1954.
 G – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – mot. perform.
 – psychol. perform. – *CAAAL-0 A-0573.

A series of controlled experiments on humans were carried out to test the effectiveness of "elektrovit" and "polysan" (a homeopathic drug prepared from various types of grasses grown in special pathogenic regions under strictly controlled conditions) as sobering agents. The subjects, having abstained from drinking 24 hr prior to the test, were given 1 1/4 l white wine after being subjected to electrovit treatment (consisting of an 4.5 volt flashlight battery with 3 cells, at 1.5 volts each in a chromed metal case). In the following test, polysan was administered po in a dose of 20 drops with a little water 10 min prior to ingestion of 1 1/4 l wine. The results of the tests (plotted graphically) show no influence of electrovit or polysan on the blood alcohol level or psychomotor performance. No mention is made of the chemical composition or pharmacological properties of polysan.

187. Burbridge, T. N., Tipton, D. L., Jr., Sutherland, V. C., and Simon, A.
EFFECT OF CHLORPROMAZINE ON BLOOD ALCOHOL LEVEL.
 Fed. Proc. (Bethesda), 17(1): 355 (O ref.), 1958.
 E – abst. – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin.
 – in vivo – blood lev. – absorp., distrib., stor. – barbiturates – tranquilizers – *CAAAL-0
 A-0574.

To analyze the cause of an increased blood alcohol level after chlorpromazine administration to alcoholics, rabbits were given first alcohol (2 g/kg of 20% ethanol), and then 3 mg/kg chlorpromazine twice daily for a week. The mean blood levels for untreated and treated animals were 1.04 and 1.50 mg/ml, respectively, at 15 min, and 1.22 and 1.50 mg/ml, respectively, at 49 min. Reserpine, phenobarbital, and saline did not have this effect, but a single dose of chlorpromazine caused the same effect.

188. Burger, E.
EINFLUSS VON TRANQUILLIZER-SUBSTANZEN AUF DIE ALKOHOLWIRKUNG.
 [Influence of tranquilizers on the action of alcohol].
 Hefte Unfallheilk. (Berlin), 66: 99-102 (3 ref.), 1961.
 G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – cardiovasc. – CNS – nerv. syst. – senses – sed., hypnot. – tranquilizers – *CAAAL-9724-J1
 A-0575.

Experiments with four students, accustomed to alcohol, are described. The effects of tranquilizers (normally prescribed daily dose) and alcohol (attaining a blood level from 1.3-1.6°/oo) upon mental and motor performance are presented. The tranquilizers included the phenothiazine derivatives, reserpine, and meprobamate. Pecazine, promazine, and meprobamate had no potentiating effects on reaction time; prothipendyl and chlorprothixene had marked potentiating effects on speech and movements 1 hr after alcohol, but not always on reaction time. Rivasin had a marked potentiating effect, and phasein-forte a slight effect. It is concluded that synergism of alcohol and tranquilizers of the prothipendyl and thiaxanthene series can detrimentally affect driving.

189. Burger, E.
BEEINFLUSSUNG DER ALKOHOLWIRKUNG DURCH LIBRIUM IM RAHMEN DES STRASSENVERKEHRS. [Influence of librium on the effect of alcohol from the viewpoint of road traffic].
 Hefte Unfallheilk. (Berlin), 75: 256-258 (O ref.), 1963.
 G – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – mot. perform. – CNS – nerv. syst. – skel., muscle, skin – tranquilizers – *CAAAL-0
 A-0576.

Five male and two female students received enough alcohol to achieve a blood alcohol level of 0.9 to 1.0°/oo. Librium was given in a dose of 1 mg/kg. The subjects were tested with librium alone, alcohol alone, and both combined. Librium did not potentiate the effects of alcohol; the combined effect was strictly additive. Librium is considered to be less of a hazard in combination with alcohol than other tranquilizers, although fatigue occurs more quickly and is more pronounced, and atactic disturbances are also found.

190. Buris, L.
CHANGES OF THE QUOTIENT URINE ALCOHOL: BLOOD ALCOHOL IN VARIOUS INJURIES AND INTOXICATIONS.
 Acta Morph. Acad. Sci. Hung. (Budapest), Suppl. 8: 61 (O ref.), 1959.
 E – SEC – abst. – exp. – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – in vitro – blood lev. – other drug lev. – CNS – liver, kidney – barbiturates – *CAAAL-0
 A-1266.

Changes in the urine alcohol: blood alcohol quotient after various injuries and drug intoxications are discussed. In cases of injuries and poisonings by narcotic compounds, it has been observed that, if the patient survived for a longer time, the urine alcohol: blood alcohol quotient increased, with values ranging from 1.7-2.34 being found instead of the physiological values 1.1-1.4. In the author's present experiments, dogs poisoned with alcohol were subjected to cerebral injury and to intoxication with the barbiturate veronal. Following cerebral injury, the urine alcohol: blood alcohol quotient did not change, whereas, in veronal poisoning, the quotient increased to 2.05 on the average. The mucosa of the urinary bladder of dogs has been found to be permeable to alcohol. In vitro, the permeability of the urinary bladder to alcohol gradually ceased. The present observations do not indicate the human bladder to be less permeable to alcohol than that of the dog.

191. Buris, L.

CHANGES OF THE URINE ALCOHOL: BLOOD ALCOHOL QUOTIENT IN EXPERIMENTAL ALCOHOL-BARBITURATE POISONING.

Acta Morph. Acad. Sci. Hung. (Budapest), Suppl. 9: 52 (0 ref.), 1960.
 E – abst. – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – liver, kidney – barbiturates – *CAAAL-0 A-1267.

The effect of experimental alcohol-barbiturate poisoning on the urine alcohol: blood alcohol quotient was studied in dogs. The urinary bladder was isolated from the kidneys by cutting the ureters, suturing the proximal stumps to the abdominal skin, and ligating the distal stumps. The bladder was then filled with 50 ml of 1-2% alcohol. Samples were taken from the bladder at 1 hr intervals, and the alcohol content was estimated by Widmark's method. The alcohol content of the bladder decreased to zero in 3 hr. When the above procedure was preceded by alcohol poisoning, the disappearance of alcohol from the bladder took 6-8 hr, and, after alcohol-barbiturate intoxication, there was alcohol in the bladder even after 24 hr. It is concluded that the permeability of the bladder wall to alcohol is greatly impaired by alcohol poisoning, and still more by alcohol-barbiturate intoxication, and that this fact accounts for the increased urine alcohol: blood alcohol quotient under such circumstances.

192. Buris, L.

ÜBER DEN QUOTIENTEN ALKOHOLGEHALT IM HARN ZU ALKOHOLGEHALT IM BLUT (ALKOHOLGEHALT IN BLUT UND LIQUOR) BEI VERSCHIEDENEN VERLETZUNGEN UND VERGIFTUNGEN. [On the quotient of alcohol content in urine to alcohol content in the blood (alcohol content in blood and fluid) in various injuries and poisonings].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 62: 221-231 (10 ref.), 1968.
 G – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – liver, kidney – barbiturates – *CAAAL-0 B-0258.

Experiments were carried out in animals and humans to determine the differences in the urine alcohol: blood alcohol quotients. In controlled experiments with dogs weighing 8 to 15 kg, 0.07 g/kg sodium veronal was given iv and ip, alcohol (iv drop-infusion) was administered in a dose of 0.5 g/kg, and the urine: blood alcohol quotient was determined 2.6 and 24 hr after infusion using Widmark's method. The results indicated that the elevated urine: blood alcohol quotient varies between 1.7 and 2.4, with a mean value of 2.0. In the control test, in which alcohol alone was used, the quotient values were not raised—6 hr after administration, the quotients were between 0.47 and 1.06, with a mean value of 0.9. The author concludes that, in the case of simultaneous alcohol-barbiturate poisoning in the state of unconsciousness, the permeability of the bladder wall undergoes a change.

193. Burkitt, R. J.

MISCELLANEOUS CASES.

Dublin Medical Press (Dublin), 1(17): 259-262 (0 ref.),

1839.

E – general – case hist. – DC (antidotal) – humans – cardiovasc. – CNS – senses – elect., water-bal. agents – integ. syst. agents – *CAAAL-0 A-0577.

The author presents 4 miscellaneous case histories, the fourth of which is a case of poisoning by alcohol. A male, aged 30, was admitted to the hospital at 1:00 p.m., unconscious, breathing stertorously, face swollen and red, pupils strongly contracted and insensitive to light, and pulse almost imperceptible. His stomach was pumped out, and he was given 2 oz camphor mixture with 10 grains ammonium carbonate (injection), bled, and a turpentine enema was given by rectum. At 6:30 p.m., 6 oz camphor mixture (1 oz/hr) was administered. At 9:30 p.m., calomel was also given. The next morning, the patient regained his senses, and, in a few days he was dismissed.

194. Burnam, W., and Erickson, C. K.
 ANTAGONISM OF ETHANOL SLEEP TIME BY CENTRAL CHOLINERGICS.
 Fed. Proc. (Bethesda), 28(2): 262 (0 ref.), 1969.
 E – abst. – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – CNS – nerv. syst. – autonomic agents – musculoskel. agents – *CAAAL-0 B-0259.

The hypothesis that the depressant action of ethanol results from antagonism of a central cholinergic system was tested in sleeping time studies on mice. A dose of 0.01 mg/kg physostigmine injected 5 min after or simultaneously with ethanol had little effect, but when injected 5 min before ethanol, a large sleeping time decrease ($p < 0.001$) was shown—from 96.9 ± 5.2 min to 73.4 ± 4.0 min. 0.5 mg/kg physostigmine produced death in most mice when injected with ethanol as above. Pretreatment with 1 mg/kg atropine or methyl atropine did not alter the effect of physostigmine or ethanol sleep time, but it did protect against the toxicity of 0.5 mg/kg ethanol. The findings point to the assumption that increased methyl atropine can overcome the central depressant action of ethanol.

195. Burner, M.
 DES EFFETS HABITUELS DE L'ALCOOLISATION AIGUE AUX MANIFESTATIONS PSYCHIATRIQUES DE L'INTOXICATION AIGUE: TOLÉRANCE ET INTOLÉRANCE À L'ALCOOL ET INFLUENCE DE CERTAINS MÉDICAMENTS. [The habitual effects of acute alcoholism on the psychiatric manifestations of acute intoxication: tolerance and intolerance to alcohol and the influence of certain drugs].
 Revue de l'Alcoolisme (Nantes), 13(4): 259-282 (59 ref.), 1967.
 F – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – DC (sensit.) – humans – CNS – metab. proc. – tranquilizers – *CAAAL-0 B-0260.

A review of literature is given on interactions between alcohol and drugs. Reference is made to experiments in which the action of chlorpromazine, reserpine and butyrophenone was increased 6, 2, and 10 times, respectively, in the 4 species of animals tested under alcohol conditions. Case material is cited in which the intake of a moderate dose of a psychopharmacological drug, with a meprobamate base, together with alcohol resulted in a state of severe shock. The author points out that acute alcoholism potentiates the effects of some neuroleptics or tranquilizers, a reaction which in some subjects may lead to a comatose state. This condition has been observed in both animals and man.

196. Burrows, E. H.
 ALCOHOL-BARBITURATE SYNERGISM.
 S. Afr. Med. J. (Capetown), 27: 1057-1059 (10 ref.), 1953.
 E – general – review – DC (add., infra-add., unspec. incr.) – med.-leg. – drug-dep. humans – mammals – acute admin. – in vivo – blood lev. – other drug lev. – CNS – respir. – barbiturates – *CAAAL-6867-D1 A-0578.

Two case histories of alcohol-barbiturate synergism are given. A woman was found dead after a severe bout of drinking, during which tuinal was taken. Brain alcohol was estimated at 0.04%, barbiturate

content was determined in the liver at 10.91 mg, the kidneys at 1.79 mg, and the stomach at 1.45 mg. A man was found dead after heavy drinking and after having taken barbiturates; he was known to be in the habit of drinking a full bottle of spirits and tapering off his hangover with tuinal capsules. Experimental and clinical evidence of an additive, and possibly potentiative, synergism between ethanol and barbiturates is reviewed.

197. Buttle, G. A. H., Fearn, H. J., and Hodges, J. R.
ALCOHOL AND BARBITURATES.
Brit. Med. J. (London), 2: 222 (2 ref.), 1953.
E – exp. cont. – general – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo
– dose resp. – barbiturates – *CAAAL-6699-D2 A-0579.

Experiments on mice were performed to determine the acute toxicity of sodium amytal (20 mg/ml) and alcohol (50 v/v in water) administered simultaneously. The results show that there is a synergism between the two drugs, but the effects are simply additive; the alcohol did not potentiate the barbiturate. Several LD₅₀ figures of different combinations of ethanol and sodium amytal are presented.

198. Büttner, H., and Portwich, F.
WIRKUNGEN DES N-(4-METHYLBENZOL-SULFONYL)-N'-BUTYL-HARNSTOFFS (D 860) AUF DEN STOFFWECHSEL DES ÄTHANOLS. [Effects of N-(4-methylbenzene-sulfonyl)-N'-butyl-urea (D 860) on the metabolism of ethanol].
Naunyn-Schmiedeberg's Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 238: 45-46 (0 ref.), 1960.
G – abst. – exp. comp. – DC (sensit.) – humans – acute admin. – in vivo – blood lev. – other drug lev. – liver, kidney – metab. proc. – hormones, hormone antag. – *CAAAL-9860-B2 A-1378.

Because several cases of intolerance to alcohol occurred during treatment of diabetic patients with D 860 (tolbutamide), an experiment was conducted on diabetic and normal humans. When tolbutamide was given in combination with 5 g/kg alcohol, a disulfiram-like reaction occurred in both groups. A significant rise in blood acetaldehyde, determined enzymatically, was found in all subjects, yet the blood and breath acetaldehyde levels and the metabolic rate of alcohol were the same before and after tolbutamide administration. Using the method of Jedeikin and Weinhouse, it was observed that, when the DPNH—the reduced form of diphosphopyridine nucleotide (DPN)—level in the liver increased, the DPN content decreased. It is concluded that the decrease in the liver DPN/DPNH ratio is due to a retarding of DPNH reoxidation. However, the question why the disturbance in DPNH reoxidation affects acetaldehyde oxidation, and not the DPN-dependent ethanol oxidation, remains to be answered.

199. Büttner, H.
ARZNEIMITTEL UND ALKOHOL. [Medicaments and alcohol].
Ther. Gegenw. (Berlin), 99(8): 384-386 (1 ref.), 1960.
G – general – DC (sensit.) – humans – blood lev. – blood comp., sites, lymph – metab. proc. – barbiturates – miscellaneous – unclass. ther. agents – *CAAAL-9892-D3 A-0580.

Some 20 to 30 compounds are known to cause an intolerance reaction if combined with alcohol. In this paper they are divided into three groups according to their reactions: drugs which increase the methemoglobin content of the blood (aromatic nitrogen compounds, e.g., aniline, aminophenol, and nitrobenzene), drugs which increase the acetaldehyde content of the blood (calcium cyanamide, disulfiram, sulfonylureas, animal charcoal, butyraldoxime, and various pyrazole derivatives), and drugs with atypical effects (barbiturates, isoniazid, and various rhodanates). The reactions, the symptoms, and the treatment are discussed.

200. Büttner, H.
 ÄTHANOLUNVERTRÄGLICHKEIT BEIM MENSCHEN NACH
 SULFONYLHARNSTOFFEN. [Intolerance to ethanol in humans after sulfonylurea intake].
 Deutsches Archiv für Klinische Medizin (Munich), 207: 1-18 (83 ref.), 1961.
 G – exp. cont. – DC (sensit.) – humans – acute admin. – in vivo – blood lev. – cardiovasc. – CNS
 – metab. proc. – respir. – hormones, hormone antag. – *CAAAL-9860-B2 A-1330.

The alcohol intolerance-inducing effects of tolbutamide (D 860) were studied. The experiments were performed in 2 phases on healthy and diabetic humans of both sexes. In the first (preliminary) phase, blood samples were taken and 0.5 g/kg alcohol was given po. Then, 10, 20, 30, 45, and 60 min after the intake of alcohol, blood tests were again performed. In the next (main) phase, 1-2 days following the preliminary phase, D 860 was given for 2 days (2-0.5 g tablets 3 times/day). 1 hr after the last dose of D 860, blood tests were made and 0.5 g/kg alcohol was administered. The effects of D 860 pretreatment (culminating 30 min, and subsiding 60 min after ethanol administration) showed as: flushing, tachycardia, tachypnea, feeling of heat, faster pulse, and falling blood pressure. The after-effects were: exhaustion, vomiting, and nausea. The blood acetaldehyde level was significantly increased. The author concludes that the observed symptoms were similar to that of the antabuse-alcohol reaction, and are possibly due to the inhibiting effect of D 860 on acetaldehyde metabolism.

201. Büttner, H., Portwich, F., and Engelhardt, K.
 DER DPN⁺-UND DPN-H-GEHALT DER RATTENLEBER WÄHREND DES ABBAUES
 VON ÄTHANOL UND SEINE BEEINFLUSSUNG DURCH SULFONYLHARNSTOFF
 UND DISULFIRAM. [The DPN⁺ and DPN-H content of the rat liver during the catabolism of
 ethanol, and its modification by sulfonylurea and disulfiram].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 240:
 573-583 (37 ref.), 1961.
 G – exp. cont. – exp. comp. – DC (sensit.) – mammals – acute admin. – chronic admin. – in vivo
 – liver, kidney – metab. proc. – *CAAAL-10353-A2 A-1379.

The effects of D-860 (tolbutamide) and disulfiram pretreatment on diphosphopyridine nucleotide (DPN) and DPNH (the reduced form of DPN) contents after alcohol administration were determined in homogenized rat livers. 1 group of rats (180-300 g) was pretreated with 25 mg tolbutamide po/day for 6-8 days, following which 1.9 g/kg alcohol in a 50% sol was administered po. Another group received 750 mg disulfiram/kg, 12 hr before the above alcohol dose. Control groups received no treatment, alcohol alone, tolbutamide alone, or disulfiram alone. 30 min after alcohol administration, the animals were decapitated, and DPN and DPNH levels were measured in the homogenized livers. In contrast to the disulfiram + alcohol group, in which no significant effect on DPN and DPNH levels was observed, there was an increase of DPNH and a simultaneous decrease of DPN in the tolbutamide + alcohol group. DPN and DPNH concentrations, expressed in $\mu\text{g/g}$ fresh liver, changed from 373 and 291 respectively, in untreated controls, to 389 and 335 after alcohol alone, to 457 and 328 after tolbutamide alone, to 343 and 403 after tolbutamide + alcohol, to 428 and 310 after disulfiram alone, and to 399 and 373 after disulfiram + alcohol. The changes in DPN and DPNH concentrations after tolbutamide + alcohol are in accordance with previously-observed changes in pyruvate and lactate contents of blood and liver under the influence of sulfonylureas.

202. Butzengeiger, K. H.
 ÜBER PERIPHERE ZIRKULATIONSTÖRUNGEN BEI CHRONISCHER
 ARSENVERGIFTUNG. [Peripheral circulatory disturbances in chronic arsenic poisoning].
 Klin. Wschr. (Berlin), 19(22): 523-527 (20 ref.), 1940.
 G – SEC – DC (unchanged) – humans – cardiovasc. – unclass. ther. agents – *CAAAL-0
 A-0581.

The paper presents case histories of 15 (out of a total of 180) vine dressers and coopers who were hospitalized because of chronic arsenic poisoning. All had developed peripheral circulatory disturbances. Six of the 15 patients suffered from gangrene in the extremities. The incidence of poisoning was due to working with insecticides. On the basis of these and other reported observations, the author considers that the peripheral circulatory disturbances (endartitis) were a direct result of the chronic arsenic poisoning, and that alcohol had no direct effect. However, it must be pointed out that arsenic was also contained in the wine which the vine dressers drank.

203. Cabana, B. E., and Gessner, P. K.

THE KINETICS OF CHLORAL HYDRATE METABOLISM IN MICE AND THE EFFECT THEREON OF ETHANOL.

J. Pharmacol. Exp. Ther. (Baltimore), 174(2): 260-275 (42 ref.), 1970.

E – exp. comp. – DC (add., infra-add., unsp. incr.) – mammals – acute admin. – in vivo – CNS – metab. proc. – sed., hypnot. – *CAAAL-0 B-0569.

The kinetics of chloral hydrate (CH) metabolism and the effects of ethanol (E) interaction were investigated in mice. The levels of CH, free trichloroethanol (TE), total TE (i.e., TE determined after hydrolysis of urochloric acid), and trichloroacetic acid (TA) were determined after administration of 500 mg/kg CH, alone or in combination with an equimolar amount of E. The degree of accumulation of TE in vivo after CH administration was predicted quantitatively and confirmed. TA formation from CH occurred at a rate of 0.0064 min^{-1} , resulting in the oxidation of 11% of administered CH. 9.6% of administered CH escaped metabolism altogether. Co-administration of E significantly increased CH disappearance to 0.075 min^{-1} ; the rate constant for TA formation was changed significantly, but significantly less TA was formed from the CH. The rate constant for TE formation was increased by 84% by co-administration of E, but the rate of TE disappearance was unchanged. It was found possible to predict a greater accumulation of TE in vivo and confirm this experimentally. After iv administration, TE was 1.18 times as potent as CH on a molar basis in causing loss of righting reflex. It is concluded that E potentiation of the effects of CH can be explained by a greater accumulation of TE.

204. Cade, J. F. J.

THE USE OF PARALDEHYDE IN ALCOHOLIC DELIRIUM TREMENS.

Med. J. Aust. (Sydney), 40(2): 276-277 (2 ref.), 1953.

E – general – DC (unspec.) – drug-dep. humans – cardiovasc. – CNS – respir. – sed., hypnot. – *CAAAL-6848-N6 A-0582.

The author replies to a paper by Steyn, Douw G. (The use of paraldehyde in alcoholic delirium tremens, Med. J. Aust. (Sydney), 40: 90-91, 1953) in which the dangers of the use of paraldehyde in treating delirium tremens (including the facts that alcoholics are more susceptible to the toxic action of paraldehyde, and that paraldehyde and alcohol act synergistically) are pointed out. He considers it the drug of choice in delirium tremens, in doses of 4 drachms po, in conjunction with chloral hydrate and potassium bromide; formerly this mixture was administered with 2 oz brandy for the initial dose, and 1 oz brandy for repeat doses.

205. Caffi, M., and Paganoni, C.

AZIONE DELLA VITAMINA B₁₂ E DI UN SUO ANALOGO NELLE NEURITI TOSSICHE ALCOOLICO-TABAGICHE (STUDIO ISTOPATOLOGICO). [Action of vitamin B₁₂ and of one of its analogues in toxic alcohol-tobacco neuritis (histopathological study)].

Ann. Ottal. (Parma), 86: 87-97 (25 ref.), 1960.

I – exp. cont. – DC (decrease) – DC (add., infra-add., unsp. incr.) – mammals – acute admin. – in vivo – CNS – metab. proc. – senses – nutritive agents – *CAAAL-9496-G2 A-0583.

Guinea pigs were divided into 4 groups. Group 1 was treated with sc injections of 1% nicotine sol, 1 cc per day. Group 2 received 30% alcohol po, 20 to 30 cc daily. Group 3 received both nicotine and alcohol. Group 4 served as control. The animals were killed after 80 days and their optic nerves were studied. The degenerative changes were more pronounced in group 3. Half of the animals in the first 3 groups were also treated with vitamin B₁₂ or C₅₆ (a synthetic analog of B₁₂). In these animals the degenerative changes were considerably less.

206. Caird, W. K., Sloane, R. B., and Inglis, J.
THE EFFECTS OF NIALAMIDE AND ETHYL ALCOHOL ON SOME PERSONALITY, COGNITIVE AND PSYCHOMOTOR VARIABLES IN NORMAL VOLUNTEERS.
Journal of Neuropsychiatry (Chicago), 2: 31-34 (8 ref.), 1960.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – acute admin. – in vivo – mot. perform. – psychol. perform. – CNS – antidepressants – *CAAAL-9499-J1
 A-0584.

The synergistic effects of nialamide with alcohol were tested. Five subjects were used in each of 2 experiments. Subjects in experiment 1 received, on 5 consecutive days, a different combination of placebo, nialamide and alcohol. The doses were 50 mg nialamide and 3 oz of 40% vodka. Subjects in experiment 2 received 150 mg nialamide and 4 oz of vodka. Different psychometric tests were given. Analysis of test results with either dose level failed to show any significant effect of nialamide or alcohol on test results, whether taken separately or together. However, the behavior of the subjects was markedly at variance with the test scores. No change in behaviour was noted after nialamide alone; in some cases intoxication occurred after alcohol alone (3-4 oz); and, when nialamide and alcohol were given together, the intoxication seemed to be more pronounced.

207. Calesnick, E.
ANTIHYPERTENSIVE ACTION OF THE ANTIMICROBIAL AGENT FURAZOLIDONE.
Amer. J. Med. Sci. (Philadelphia), 236: 736-746 (10 ref.), 1958.
 E – exp. – DC (sensit.) – humans – acute admin. – in vivo – CNS – metab. proc. – skel., muscle, skin – anti-infectants – unclass. ther. agents – *CAAAL-0
 A-1268.

A sensitization reaction to alcohol produced by the antimicrobial agent furazolidone is reported. The reaction was observed in a male patient who was among 16 hypertensive subjects participating in a clinical evaluation of the antihypertensive action of the drug. The subjects had all been started on a po dosage of 800 mg furazolidone/day, and this dosage was gradually reduced. The reaction occurred within 10 min of ingesting some beer, and was characterized by facial flushing, lacrimation, conjunctivitis, weakness, and light-headedness. These symptoms appeared every time the subject drank beer, and persisted for 20-45 min. The author comments that the symptoms observed were similar to those produced by alcohol in an individual sensitized by disulfiram. This suggests that furazolidone, like disulfiram, may inhibit in vivo the enzyme system responsible for the conversion of acetaldehyde to acetate, thereby producing an elevated blood acetaldehyde level.

208. Campana, C.
ANTIDIABETICI ORALI ED ALCOOL. [Oral anti-diabetics and alcohol].
Polislinico, Sezione Pratica (Rome), 68: 221-222 (0 ref.), 1961.
 I – general – DC (sensit.) – humans – cardiovasc. – metab. proc. – hormones, hormone antag. – *CAAAL-0
 A-1380.

A question is asked concerning a diabetic patient undergoing treatment with antidiabetic sulfonamides, who experiences heat sensations and flushing of the face at mealtimes; these reactions do not appear if the drugs are not taken. In reply, it is noted that these cutaneous allergic phenomena can occur during therapy with synthetic antidiabetic agents, including tolbutamide, chlorpropamide, and,

in particular, carbutamide. When taken in conjunction with alcohol, these and other sulfonylureas, especially in large doses, can have an effect similar to that of antabuse, causing an increase in the level of acetaldehyde in the blood, with circulatory reactions, breathing and gastroenteric difficulties, tachycardia, cutaneous reddening, arterial hypotension, hyperpnea, nausea or vomiting, and general malaise. In the case described, the patient should attempt at least 1 meal without wine or other alcoholic beverages.

209. Canessa, I., Valiente, S., and Mella, I.

CLINICAL EVALUATION OF CHLORPROPAMIDE IN DIABETES MELLITUS.

Ann. N.Y. Acad. Sci. (New York), 74: 752-770 (1 ref.), 1958-59.
 E – SEC – general – DC (sensit.) – humans – chronic admin. – in vivo – blood lev. – cardiovasc. – CNS – G.I. tract – metab. proc. – respir. – skel., muscle, skin – hormones, hormone antag. – unclass. ther. agents – *CAAAL-0 A-1269.

An intense reaction with alcohol was experienced by 10 patients participating in a clinical study of chlorpropamide therapy in 80 diabetics. The subjects, who were maintained on 150-250 g of carbohydrates and 0.125-1.0 g chlorpropamide/day, were observed from 30-210 days. In 2 cases the reaction was produced deliberately, and observed to be identical to that described for the other sulfonylureas and for tetraethylthiuram disulfide (antabuse). It was characterized by a reddening of the face, feeling of anguish, dyspnea, palpitations, and, more rarely, vomiting. The reaction was extremely violent and prolonged in 1 case, whereas, in other patients who took very moderate amounts of alcohol (wine) at meals, the reaction did not occur, was only mild, or eventually ceased to occur. The authors interpret the reaction as a consequence of blocking the oxidation of alcohol in the acetaldehyde stage, and suggest that patients receiving chlorpropamide should be prevented from consuming any alcohol whatsoever.

210. Cardonnet, L. J., Staffieri, J. J., Eberhardt, D. R., Tommasino, P. O., Berli, R. R., and Muratorio, J.

CLINICAL STUDY OF THE EFFECTS OF CHLOROPROPAMIDE ON NORMAL SUBJECTS AND ON DIABETICS.

Ann. N.Y. Acad. Sci. (New York), 74: 771-787 (16 ref.), 1958-59.
 E – SEC – general – DC (sensit.) – humans – chronic admin. – in vivo – skel., muscle, skin – hormones, hormone antag. – *CAAAL-0 A-1270.

A sensitization reaction to alcohol produced by the oral hypoglycemic drug chlorpropamide is reported. The reaction was observed during a clinical evaluation of the drug involving 60 diabetic and 30 non-diabetic subjects. A group of 10 diabetics was administered 1.5 g of chlorpropamide/day for 2 days, followed by 1 g/day for not less than 15 days. Of this group, 9 subjects experienced a reaction of the vasomotor type, accompanied by feverishness and flushing, which occurred after the ingestion of an alcoholic beverage. The reaction persisted for about 30 min, and did not recur if the dose of the drug was reduced or the ingestion of alcohol was suspended. Untoward side-effects of this sort were not observed when the estimated effective therapeutic maintenance dosage of 250-750 mg of chlorpropamide/day was administered. It is concluded that, although no definite pathogenic mechanism is obvious, the inhibition of alcoholic dehydrogenation is a possible explanation for the reaction.

211. Carlton, P. L.

THE INTERACTING EFFECTS OF SODIUM PENTOBARBITAL AND TWO ALCOHOLS ON THE OPERANT BEHAVIOR OF THE RAT.

U.S. Army Medical Research Laboratory, Ft. Knox, Kentucky, Report No. 304, 12 pp. (4 ref.), 1957.

E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – absorp., distrib., stor. – metab. proc. – barbiturates – *CAAAL-8097-J2 A-0585.

The operant behaviour of albino rats working under a fixed-interval schedule of reinforcement was found to be disrupted by small dosages of sodium pentobarbital (10 mg/kg) in water, although these dosages produced no directly observable change in the animal. On the other hand, when the barbiturate was in a sol of 10% ethyl alcohol, 20% propylene glycol and 70% water, no change in operant response characteristics was observed. It is suggested that the effect of the 2 alcohols in the injection sol may be a function of a synaptic facilitation which they induce and which serves to compensate for the barbiturate-induced depression.

212. Carpenter, J. A.

EFFECTS OF ALCOHOLIC BEVERAGES ON SKIN CONDUCTANCE: AN EXPLORATORY STUDY.

Quart. J. Stud. Alcohol (New Haven), 18: 1-18 (13 ref.),

1957.

E – SEC – exp. cont. – congen. stud. – humans – in vivo – mot. perform. – nerv. syst. – skel., muscle, skin – *CAAAL-8264-J1 A-1381.

The effects of alcohol, in the form of 12% wine or 12% alcohol sol (in 2 amounts—50 and 350 ml), on skin conductance were measured in 8 men (all moderate social drinkers) at pre-, early-, and post-drinking times. A task of card filing was assigned, and a loud horn provided a startle stimulus. Both conductance level, assumed to represent sympathetic nervous system activity, and galvanic skin response (GSR) were measured. In the late post-drinking period, there was a significant interaction between the form of the beverages used and the dosages. At a dosage of 50 ml, alcohol was more effective in lowering conductance level than was wine, whereas, at 350 ml, wine was more effective than alcohol. The magnitude of the GSR was reduced by the 350 ml dose of beverage; wine was slightly more effective than the alcohol sol in reducing the GSR. The GSR was negatively related to the pre-drinking conductance level, indicating that some of the characteristic responsiveness of the subjects remained after alcohol ingestion; also, the GSR was negatively related to the conductance level at the time of measurement of the former. It is suggested that small but measureable changes in autonomic nervous system activity, resulting from the ingestion of a moderate amount of alcohol, may be related to the persistence and widespread nature of social drinking.

213. Carpenter, J. A.

THE EFFECTS OF CAFFEINE AND ALCOHOL ON SIMPLE VISUAL REACTION TIME.

J. Comp. Physiol. Psychol. (Washington), 52: 491-496 (11 ref.),

1959.

E – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – other drug lev. – CNS – senses – stimulants – *CAAAL-4002-J1 A-0586.

9 male subjects received 9 combinations of 2 drugs, each represented by 3 dosage levels—caffeine (doses: 0.0, 1.47 and 2.94 mg/kg) and alcohol (doses: 0.0, 0.4 and 0.8 ml/kg of absolute alcohol)—in the form of whiskey in ginger ale. Blood alcohol levels were measured with the alcometer, and two different intensities of light stimulus were used. When the differences in stimulus intensities were ignored, a highly significant positive relationship between reaction time (RT) and alcohol dose was observed; however, the magnitude of the RT difference between the zero and high alcohol dose was only 12 msec, an amount of little significance. Caffeine was effective in altering the size of the differences in RT to the high and low stimulus intensities. This suggested a 3-way interaction between alcohol, caffeine, and intensities which was not substantiated. It was tentatively concluded that, in spite of the strong relation between RT and alcohol dose (over the range of dosage administered), the magnitude of the change effected by alcohol was a relatively unimportant factor in behaviour.

214. Carpenter, J. A., and Varley, M.

THE JOINT ACTION OF TRANQUILIZERS AND ALCOHOL ON DRIVING.

In: Havard, J. D. J., ed. *Alcohol and Road Traffic*. Proceedings of the Third International Conference

on Alcohol and Road Traffic at London, September 3-7, 1962. London: British Medical Ass., pp. 156-161 (19 ref.), 1963.

E – presentation – review – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mot. vehic. – humans – acute admin. – chronic admin. – in vivo – dose resp. – blood lev. – mot. perform. – psychol. perform. – CNS – tranquilizers – *CAAAL-0 A-0587.

Reviewed are the experimental papers on driving when alcohol, tranquilizers, or both are used. Impairment occurs at blood alcohol levels near 0.05%. Severe behavioural deterioration after small amounts of alcohol and chronic administration of meprobamate is reported. Problems in the interpretation of results are discussed.

215. Carpenter, R. K., and MacLeod, L. D.
THE EFFECTS OF ETHYL ALCOHOL AND ACETALDEHYDE ON MAZE BEHAVIOUR AND MOTOR CO-ORDINATION IN RATS.
Journal of Mental Science (London), 98: 167-173 (18 ref.), 1952.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – mot. perform. – cardiovasc. – CNS – metab. proc. – *CAAAL-6062-J2 A-0588.

Alteration of maze behaviour and motor-co-ordination were studied to test the degree of intoxication in rats. 19 male and 19 female rats made 6 runs before and after administration of 4 ml of 30% alcohol/160 g by stomach tube. The same test was applied after administration of alcohol, acetaldehyde, and alcohol and acetaldehyde together. The effect of acetaldehyde alone was short; the effect of acetaldehyde and alcohol in combination was much greater than after each drug alone. Detailed results are shown in tables.

216. Carpenter, T. M.
THE METABOLISM OF ALCOHOL: A REVIEW.
Quart. J. Stud. Alcohol (New Haven), 1(2): 201-226 (166 ref.), 1940.
E – SEC – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – metab. proc. – analg., antipyret. – autonomic agents – hormones, hormone antag. – neoplast. agents – stimulants – *CAAAL-23-A1 A-0589.

The results of previous experiments are reviewed with respect to the influence of drugs and hormones on metabolism of alcohol. In dogs given 7.5-15 mg/kg dinitrophenol, the disappearance of alcohol was greatly increased when no hyperthermia was produced. Caffeine and atropine had no effect on metabolism, but the intoxicating effect was lowered by 0.2 g caffeine. Morphine caused a lower alcohol curve by 20-40%, and urethane caused a reduction in rate of oxidation in rats and rabbits of 30%. Generally, when large doses of insulin were given to humans, there was some increase in rate of disappearance. Other drugs studied which normally had no effect were thyroxine, adrenaline, and pituitrin.

217. Carratalá, R.
LA ETILOTERAPIA EN LAS INTOXICACIONES DETERMINADAS POR LOS BARBITÚRICOS Y POR EL ALCOHOL. [Ethanol therapy in poisonings due to barbiturates and alcohol].
Revista de Psiquiatria y Criminologia (Buenos Aires), 6: 297-306 (4 ref.), 1941.
Sp – exp. – DC (antidotal) – DC (decrease) – humans – mammals – acute admin. – in vivo – CNS – barbiturates – *CAAAL-0 A-1331.

Two separate studies are described—the treatment of barbiturate poisoning, and the treatment of manic ethylism with 30% ethanol iv. Experiments were conducted on rabbits and dogs in which

veronal (0.550 g/kg in rabbits), luminal (0.150 g/kg in rabbits, 0.300 g/kg in dogs), and dial (0.120 or 0.180 g/kg in rabbits) were administered iv 5 min after 30% ethanol (2-3 cc in rabbits, 3 cc in dogs); the 3 barbiturates were also given in the same dosages, followed by 15-18 cc 30% ethanol. It was found that ethanol delays the death of animals subsequently poisoned by barbiturates. Used as an antidote, it defers the beginning of coma, though it does not always prevent death. It is impossible to foretell the efficiency of ethanol therapy for barbiturate poisoning in man, and in acute states of poisoning it is always dangerous. The authors suggest treatment with 5-30 cc coramine/24 hr or 3 cc doses of cardiazol (not to exceed 15-30 cc/day) for mild poisoning, and strychnine (1 cg/hr) and picrotoxin (1:1000 sol, 1 cc/min iv) in severe poisonings. Experiments on, and treatment of manic ethylism are also discussed.

218. Carratalá, R.
LA ETILOTERAPIA EN LAS INTOXICACIONES DETERMINADAS POR LOS BARBITÚRICOS Y POR EL ALCOHOL. [Ethanol therapy in poisonings due to barbiturates and alcohol].
 Revista de Psiquiatria y Criminologia (Buenos Aires), 6: 656-657 (0 ref.), 1941.
 Sp – abst. – exp. – DC (antidotal) – DC (decrease) – humans – mammals – acute admin. – in vivo – CNS – barbiturates – *CAAAL-3568-N7 A-0590.

Experiments on, and treatment of, barbiturate poisoning and manic ethylism are described. In dogs and rabbits, coma resulting from barbiturates was delayed if alcohol was given previously. The author considers that for man, ethanol therapy for acute barbituric intoxication is ineffective and may be dangerous. In cases of intoxication resulting from small doses of barbiturates, coramine, coranodia, and cardiazol are recommended; in poisoning from large doses, strychnine or picrotoxin should be used.

219. Carratalá, R.
LA ETILOTERAPIA EN LAS INTOXICACIONES DETERMINADAS POR LOS BARBITÚRICOS Y POR EL ALCOHOL. [Ethanol therapy in poisonings due to barbiturates and alcohol].
 Revista Medica Municipal (Rio de Janeiro), 3: 247-258 (4 ref.), 1942.
 P – exp. – DC (antidotal) – DC (decrease) – humans – mammals – acute admin. – in vivo – CNS – barbiturates – *CAAAL-0 A-0591.

Two separate studies are described—the treatment of barbiturate poisoning, and the treatment of manic ethylism with 30% ethanol iv. Experiments were conducted on rabbits and dogs in which veronal (0.550 g/kg in rabbits), luminal (0.150 g/kg in rabbits, 0.300 g/kg in dogs), and dial (0.120 or 0.180 g/kg in rabbits) were administered iv 5 min after 30% ethanol (2-3 cc in rabbits, 3 cc in dogs); the 3 barbiturates were also given in the same dosages, followed by 15-18 cc 30% ethanol. It was found that ethanol delays the death of animals subsequently poisoned by barbiturates. Used as an antidote, it defers the beginning of coma, though it does not always prevent death. It is impossible to foretell the efficiency of ethanol therapy for barbiturate poisoning in man, and in acute states of poisoning it is always dangerous. The authors suggest treatment with 5-30 cc coramine/24 hr or 3 cc doses of cardiazol (not to exceed 15-30 cc/day) for mild poisoning, and strychnine (1 cg/hr) and picrotoxin (1:1000 sol, 1 cc/min iv) in severe poisonings. Experiments on, and treatment of manic ethylism are also discussed.

220. Carratalá, R.
LA BENZEDRINA FRENTE A LA ACCIÓN DEL ALCOHOL. [Benzedrine antagonism of the action of alcohol].
 Revista de Psiquiatria y Criminologia (Buenos Aires), 10: 63-70 (3 ref.), 1945.
 Sp – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – species or sex diff. – CNS – amphetamines – *CAAAL-0 A-0592.

The action of benzedrine on the autonomic control centers and the effect of alcohol on the central nervous system were investigated in rabbits, dogs, and mice. The minimum lethal doses were, for benzedrine: rabbit, 0.03 g/kg, dog, 0.026 g/kg, mouse, 0.023 g/kg; and for 95% alcohol: rabbit, 8.65 cc/kg, dog, 7.25 cc/kg, mouse, 5.35 cc/kg. In simultaneous administration, benzedrine was antagonistic to the effects of alcohol.

221. Carratalá, R.
ALCOHOL Y ANESTESIA. [Alcohol and anesthesia].
Archivos de Medicina Legale (Buenos Aires), 18: 183-199 (0 ref.), 1948.
Sp – FS – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
– chronic admin. – in vivo – dose resp. – blood lev. – cardiovasc. – CNS – respir. – anesthetics –
barbiturates – *CAAAL-5154-D3 A-0593.

The effect of alcohol on the toxicities of various anesthetics (chloroform, ether, novocaine, and barbiturates) was investigated in dogs and rabbits. The minimum lethal dose of alcohol, administered by gastric and iv routes, and stages of intoxication in terms of blood alcohol levels, were determined. The anesthetic doses of the various drugs were determined in normal animals. The latter were compared with those in alcoholized animals. It was found that greater quantities of anesthetics are necessary to produce narcosis in the first (euphoric) stage of alcohol intoxication.

222. Carratalá, R.
ALCOHOL Y ANESTESIA. [Alcohol and anesthesia].
Hospital (Rio de Janeiro), 35(2): 215-229 (0 ref.), 1949.
Sp – FS – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
– chronic admin. – in vivo – dose resp. – blood lev. – cardiovasc. – CNS – respir. – anesthetics –
barbiturates – *CAAAL-5154-D3 A-0594.

The effect of alcohol on the toxicities of various anesthetics (chloroform, ether, novocaine, and barbiturates) was investigated in dogs and rabbits. The minimum lethal dose of alcohol, administered by gastric and iv routes, and stages of intoxication in terms of blood alcohol levels, were determined. The anesthetic doses of the various drugs were determined in normal animals. The latter were compared with those in alcoholized animals. It was found that greater quantities of anesthetics are necessary to produce narcosis in the first (euphoric) stage of alcohol intoxication.

223. Carrière, G., and Huriez, C.
LE COMA BARBITURIQUE ET SON TRAITEMENT. [Barbiturate coma and its treatment].
Médecine (Paris), 15(1): 693-717 (5 ref.), 1934.
F – general – review – DC (antidotal) – DC (decrease) – humans – mammals – liver, kidney – respir.
– barbiturates – *CAAAL-0 A-0595.

The authors describe the etiology and pathology of barbiturism and offer experimental bases for its treatment. Strychnine (1 cg/hr iv) and coramine (3 to 5 cc/hr iv) were used as antidotes for barbiturate coma. In experiments with animals, iv injections of 1 cc/kg 30% alcohol proved capable of retarding the onset of symptoms of barbiturate poisoning. In man, iv injections of 30 to 50 cc 30% alcohol were well tolerated and did not affect hepatorenal functions. Human posology rests on the study of a few cases in which 30 cc of 30% alcohol were administered iv each hr, without surpassing 200 cc, until a definite reaction was observed. It is concluded that the combined action of the three antagonists administered iv in the stipulated doses will reduce the number of deaths caused by barbiturates.

224. Carrière, G., Huriez, C., and Willoquet, P.
LE TRAITEMENT ACTUEL DU BARBITURISME AIGU: RECHERCHES
EXPÉRIMENTALES SUR L'ANTIDOTISME GARDÉNAL-CORAMINE ET

GARDÉNAL-ALCOOL. [The present treatment of acute barbiturism: experimental studies of the antagonism of gardenal-coramine and gardenal-alcohol].

Lancette Française; Gazette des Hôpitaux Civils et Militaires (Paris), 107(41): 745-752 (3 ref.),

1934.

F – exp. comp. – case hist. – DC (antidotal) – DC (decrease) – humans – mammals – acute admin. – in vivo – CNS – liver, kidney – barbiturates – *CAAAL-0 A-0596.

A series of experiments were carried out in which rabbits were given injections of 3-14 cc 30% alcohol iv, followed 5 min later by 0.125-0.32 g/kg gardenal im. It was shown that iv injections of 30% alcohol/hr in the given dosages were able to retard the onset of barbiturate coma, effecting recovery in 5-33 hr in the experimental animals. Regarding alcohol's application to humans, the authors treated a young woman for barbiturate coma (following ingestion of 5 cg gardenal) with 30% alcohol iv (20 cc/hr). She awakened completely after the fourth injection. Results of experiments with strychnine and coramine are also tabulated.

225. Carrière, G., Huriez, C., and Willoquet, P.

ÉTUDE EXPÉRIMENTALE DES INJECTIONS INTRA VEINEUSES D'ALCOOL AU COURS D'INTOXICATIONS PAR LE GARDÉNAL. [Experimental study of intravenous injections of alcohol during gardenal intoxication].

C. R. Soc. Biol. (Paris), 116: 188-190 (0 ref.),

1934.

F – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – CNS – barbiturates – *CAAAL-0 A-0597.

The authors attempted in this study to more accurately define the reported antagonism of barbiturates and alcohol. 18 rabbits received injections of 30% alcohol (1 cc/kg body weight), and were then subjected to overdoses of gardenal, followed by more alcohol injections, or else the animals were administered gardenal overdoses and then given alcohol injections. In all test animals, a cure was effected, whereas, of the 4 control animals (2 rabbits and 2 dogs), 2 died. The authors thus conclude that iv alcohol can retard the onset and rapidly cure (5-7 hr) barbiturate coma. They are not prepared to explain the mechanism of the barbiturate-alcohol antagonism until further research has been conducted.

226. Carrière, G., and Huriez, C.

TRAITEMENT DE L'INTOXICATION AIGUE PAR LES BARBITURIQUES:

STRYCHNINE? CORAMINE? ALCOOL INTRA VEINEUX? [Tréatment of acute barbiturate intoxication: Strychnine? Coramine? Intravenous alcohol?].

Écho Médicale du Nord (Lille), 2: 178-191 (0 ref.),

1934.

F – general – review – DC (antidotal) – DC (decrease) – humans – mammals – CNS – barbiturates – *CAAAL-0 A-0598.

The authors cite the case of a 23 yr-old woman who, in a suicide attempt, ingested 5 g veronal, and, to hasten death, also took 100 cc 95% alcohol. Her subsequent recovery is attributed by the authors to the antagonistic effect of alcohol. Drawing on experimental evidence, the authors feel that iv injections of 1 cc alcohol (30%)/kg are able to retard the onset of barbituric coma in animals and effect a cure in 5 to 10 hr. Human posology is based on the study of a few cases in which 30 cc of 30% alcohol were injected iv every hr without surpassing 200 cc. The authors feel that the research is too incomplete to explain the antagonism between barbiturates and alcohol, and that the assumption of an antidotism does not imply neutralization. It is concluded that the combined actions of iv injections of strychnine (1 cc/hr), coramine (3-5 cc/hr), and alcohol (20 cc/hr) would reduce the number of deaths by ureides.

227. Carrière, G., Huriez, C., and Willoquet, P.
RÔLE DES INJECTIONS INTRAVEINEUSES D'ALCOOL À 30 P. 100 DANS LE TRAITEMENT DU BARBITURISME AIGU. [Role of intravenous injections of 30% alcohol in the treatment of acute barbiturism].
 Académie de Médecine, Bulletin (Paris), 111: 655-661 (0 ref.), 1934.
 F – exp. cont. – case hist. – DC (antidotal) – DC (decrease) – humans – mammals – acute admin. – in vivo – CNS – barbiturates – *CAAAL-0 A-0599.

The possible beneficial effects of iv alcohol in acute barbiturism was investigated. 18 rabbits were injected with gardenal (0.125-0.32 g/kg), resulting in deep coma. After repeated injections (iv) of 30% alcohol, they returned to normal. A woman who had taken 30-5 cg pills of gardenal was given an iv injection of 20 cc 30% alcohol each hour. She revived by the fourth injection. From experimentation, the human dosage is 30 cc 30% alcohol.

228. Carrière, G., and Huriez, C.
DISCUSSION CLINIQUE ET THÉRAPEUTIQUE DE ONZE CAS DE COMAS BARBITURIQUES DONT TROIS MORTELS MALGRÉ DES INJECTIONS INTRAVEINEUSES DE STRYCHNINE-CORAMINE-ALCOOL. [Clinical and therapeutic discussion of eleven cases of barbiturate comas of which three were fatal despite intravenous injections of strychnine-coramine-alcohol].
 Presse Med. (Paris), 43(24): 465-469 (2 ref.), 1935.
 F – general – case hist. – DC (antidotal) – post-mort. – humans – blood lev. – other drug lev. – blood comp., sites, lymph – cardiovasc. – CNS – liver, kidney – respir. – barbiturates – *CAAAL-0 A-0600.

A discussion is given of the treatment of 11 cases of barbiturate coma, of which 2 cases received strychnine therapy, 2 cases alcohol therapy (under the theory that alcohol is antagonistic to barbiturates), and 7 cases a combination of strychnine-coramine-alcohol therapy. Injections were given iv each hr according to the following dosages: a) 1 cg strychnine, max 30 cg, b) 3 cc coramine, max 55 cc, and c) 20 cc alcohol 30%, max 150 cc. There were three fatalities; these were considered to be due to evacuatory therapy having been given too late.

229. Carrière, G., and Huriez, C.
TRAITEMENT DE L'INTOXICATION AIGUË PAR LES BARBITURIQUES: STRYCHNINE? CORAMINE? ALCOOL INTRAVEINEUX? [Treatment of acute barbiturate intoxication: Strychnine? Coramine? Intravenous alcohol?].
 Clinique (Paris), 30: 27-29 (2 ref.), 1935.
 F – general – review – DC (antidotal) – DC (decrease) – humans – mammals – CNS – liver, kidney – barbiturates – *CAAAL-0 A-0601.

Therapeutic methods using strychnine, coramine or alcohol are compared for treating acute barbiturate intoxication. The method of iv injection of 30 to 50 cc 30% alcohol is considered the safest and fastest. The injections must be iv to avoid complications of the liver, and should not exceed 200 cc in total. A combination of strychnine, 1 cc/hr, coramine, 3-5 cc/hr, and 30% alcohol, 20 cc/hr, has been found to effectively reduce the number of deaths.

230. Carroll, R. B.
ANALYSIS OF ALCOHOLIC BEVERAGES BY GAS-LIQUID CHROMATOGRAPHY.
 Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 6-19 (20 ref.), 1970.
 E – exp. – congen. stud. – *CAAAL-12884 B-0506.

The techniques of gas-liquid chromatography and mass spectrography were applied to analysis of congener components of Scotch, Irish, Canadian, bourbon, rye, straight and blended whiskies, Ameri-

can and French apple brandy, Napoleon and new French grape brandy, rum, vodka, vermouth, and gin. The principle components identified were acetaldehyde, ethyl acetate, methanol, n-propanol, and isoamyl alcohol. The results are presented in graphic and tabular form. The total congener content of the beverages varied from as low as 3 g/100 l in vodka to 285 g/100 l in bourbon, a large part of the difference being due to ethyl acetate and isoamyl alcohol. The coupling of a gas chromatograph to a mass spectrograph allows, together with use of a computer, instant qualitative and quantitative analysis. Temperature programming coupled with the use of dual columns, with or without dual detectors, permits analysis of components with a boiling point varying from cryogenic temperatures to several hundred degrees. Such refinement of methods of analysis will allow the continual emergence of a more complete knowledge of congener beverages.

231. Carulli, N., and Manenti, F.
MICROSOMAL OXIDATION OF ETHANOL AND THE DRUG METABOLIZING SYSTEM: STUDIES IN ANIMALS AND HUMANS.
 Industr. Med. Surg. (Chicago), 39(7): 310 (0 ref.), 1970.
 E – abst. – exp. cont. – cross-tol. – humans – acute admin. – chronic admin. – blood lev. – liver, kidney – metab. proc. – hormones, hormone antag. – *CAAAL-0 B-0917.

A possible relation between hepatic microsomal ethanol oxidation and drug metabolism was investigated. Both the rate of the metabolism of drugs in alcoholics, and the rate of blood ethanol clearance in well-compensated diabetic subjects treated for not less than a year with tolbutamide only, were studied. Preliminary results have shown that drugs, such as tolbutamide, which are handled by the microsomal metabolizing system, are cleared from the blood of alcoholics almost twice as fast as in normal control subjects. Diabetics on tolbutamide showed an increased blood ethanol clearance, indicating a microsomal induction produced by tolbutamide. As well, tolbutamide half-life in these diabetic subjects was half the control value.

232. Casier, H., and Delaunois, A. L.
INFLUENCE DES DINITRODÉRIVÉS, SPÉCIALEMENT DU DINITROCRÉSOL ET DU DINITROPHÉNOL, SUR LA RÉSORPTION ET L'ÉLIMINATION DE L'ALCOOL PAR L'ORGANISME. [Influence of dinitro-derivatives, especially dinitrocresol and dinitrophenol, on the absorption and elimination of alcohol by the organism].
 Arch. Int. Pharmacodyn. (Gand), 69(2): 156-180 (100 ref.), 1943.
 F – exp. – DC (decrease) – mammals – other org. – acute admin. – in vivo – in vitro – blood lev. – other drug lev. – absorp., distrib., stor. – metab. proc. – respir. – indust. intox. – *CAAAL-4412-A2 A-0602.

Reviewed are 100 experiments on the effect of temperature on the metabolism of alcohol. Reported are in vivo experiments on dogs and guinea pigs and in vitro experiments with tissues of pigeons. Elimination of alcohol by lung was increased by dinitrocresol in dogs, and increased 19% by dinitrophenol in guinea pigs. In vitro experiments on pigeon pectoral muscle tissue showed that dinitrophenol had no significant effect on oxidation of alcohol by tissues.

233. Casier, H., Jolie, J., and Bruyneel, N.
INFLUENCE DU DINITRO-CYCLO-PENTYL-PHÉNOL SUR LA COMBUSTION, L'ÉLIMINATION ET LA FIXATION DE L'ALCOOL ÉTHYLIQUE RADIOACTIF PAR L'ORGANISME. [The effect of dinitro-cyclo-pentyl-phenol on the combustion, elimination and fixation of radioactive ethyl alcohol by the organism].
 Arch. Int. Pharmacodyn. (Gand), 104(3-4): 245-274 (20 ref.), 1956.
 F – ES – exp. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – liver, kidney – metab. proc. – respir. – *CAAAL-8114-A2 A-0603.

Immediately before administration of 0.3 cc alcohol in 33% sol ip, mice received a dose of 0.3 cc of a 0.16 g°/oo sol of dinitro-cyclo-pentyl-phenol (DPP) ip. Simultaneous administration of DPP caused a 40% faster rate of oxidation during the first half hour. When DPP was administered an hr after the alcohol, the oxidation rate was increased by 20% 1/2 hr after DPP administration. All results are presented in detail in 18 tables and 10 graphs.

234. Casier, H.

MÉTABOLISME DE L'ALCOOL ÉTHYLIQUE MARQUÉ ET INFLUENCES DE SUBSTANCES PHARMACOLOGIQUES. [Metabolism of labeled ethyl alcohol and the influence of pharmacological substances].

Bull. Schweiz. Akad. Med. Wiss. (Basel), 16: 15-24 (14 ref.), 1960.

F – ES – GS – IS – exp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – liver, kidney – metab. proc. – autonomic agents – hormones, hormone antag. – unclass. ther. agents – *CAAAL-9878-A2 A-0604.

Radioactive alcohol diluted to 1/3, acetaldehyde, and acetate (C¹⁴), were injected ip into groups of 3 mice each. 90 to 92% of the alcohol was oxidized; oxidation was greatest during the first 5 hr, diminishing gradually to the 16th hr. Adrenalin diminished the fixed substances from radioactive alcohol by 6.94% 1 hr after injection. Alcohol injected for 8 days increased the contents of fatty acids and cholesterol fourfold. Dinitro-cyclo-pentyl-phenol did not influence alcohol combustion, and disulfiram inhibited combustion.

235. Casier, H., Danechmand, L., De Schaepdryver, A., Hermans, W., and Piette, Y.

BLOOD ALCOHOL LEVELS AND PSYCHOTROPIC DRUGS.

Arzneimittelforschung (Aulendorf), 16(11): 1505-1507 (9 ref.), 1966.

E – GS – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – humans – acute admin. – chronic admin. – in vivo – blood lev. – antidepressants – tranquilizers – *CAAAL-0 B-0261.

The influence of five commonly-used psychotropic drugs on blood alcohol levels after 0.5 g/kg of ethanol was studied in 60 human volunteers. Nialamide (3 x 25 mg), chlorpromazine (3 x 50 mg), and haloperidol (2 x 1 mg) produced significantly lower blood alcohol levels 30 min after ethanol; nialamide also lowered the blood alcohol level after 60 min. No change was noted after 90 min, 3 hr, or 4 hr. Imipramine (3 x 25 mg) and diazepam (2 x 5 mg) were without influence on the blood alcohol level at any time after ethanol.

236. Castex, M. R., Camponovo, L. E., Labourt, F. E., and Firmat, J.

EFFECTO DEL ACIDO SUCCINICO EN LA INTOXICACION ALCOHOLICA. [Effect of succinic acid in alcoholic intoxication].

Prensa Med. Argent. (Buenos Aires), 38(2): 55-61 (25 ref.), 1951.

Sp – exp. – DC (antidotal) – DC (decrease) – humans – mammals – acute admin. – in vivo – blood lev. – species or sex diff. – CNS – respir. – unclass. ther. agents – *CAAAL-0 A-0605.

Experiments are reported with 10 rabbits and 7 dogs receiving alcohol and succinic acid. Succinic acid counteracted the effects of alcohol, causing a decrease in the frequency and an increase in the amplitude of the respiration record. Ten patients admitted to hospital while drunk showed blood alcohol levels between 100 and 300 mg/100 cc. The injection of 125 to 300 mg succinic acid resulted in a rapid decline of the blood alcohol levels, i.e. from 300 to 200 mg/100 cc in 15 min in one patient, and from 200 to 600 mg/100 cc in 15 min in another. The clinical state of the patients paralleled the alcohol levels. The analgesic effect of alcohol was also counteracted by succinic acid.

237. Cavalieri, U., Quadri, A., and Tammaro, A. E.
 SU UNA AZIONE RESERPINO-SIMILE DELL'ALCOOL NELL'ORGANISMO SENILE.
 [On a reserpine-like action of alcohol in the aged organism].
 G. Geront. (Florence), 10: 1355-1359 (5 ref.), 1962.
 I – ES – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – cardiovasc. – CNS – glands
 – autonomic agents – *CAAAL-0 A-0606.
- 13 Subjects between 65 and 79 yr received 20 mg ephedrine 30 min after receiving 1 mg reserpine, or 45 min after receiving 1 g/kg alcohol. Both substances inhibited the hypertensive action of ephedrine. Consequently, there seems to be a similarity of action between ethanol and reserpine.
238. Cavalieri, U., Tammaro, A. E., and Quadri, A.
 SU ALCUNI RAPPORTI TRA NIALAMIDE, RESERPINA ED ALCOOL
 NELL'ORGANISMO SENILE. [On some relations between nialamide, reserpine, and alcohol in the aged body].
 G. Geront. (Florence), 12: 623-628 (3 ref.), 1964.
 I – ES – exp. comp. – DC (unchanged) – humans – acute admin. – in vivo – cardiovasc. – autonomic
 agents – *CAAAL-11309-D1 A-0607.
- In previous publications, alcohol was found to inhibit the pressor response to ephedrine. To evaluate whether alcohol acts on the basis of a reserpine-like mechanism, the influence of alcohol and reserpine on the pressor response to norepinephrine during treatment with nialamide was studied in human subjects, aged from 61 to 74 yr. Pretreatment with reserpine increased the pressor response to norepinephrine in nialamide-treated subjects; such an increase was not observed after pretreatment with ethanol. The mechanism of action of ethanol seems to be more complex than that of reserpine.
239. Certhoux, J., and Ramet, M.
 A PROPOS DE L'ALCOOLÉMIE: INCIDENCES MÉDICO-LÉGALES. [Concerning blood
 alcohol: medicolegal implications].
 Ann. Medicopsychol. (Paris), 120/1: 359-364 (0 ref.), 1962.
 F – exp. comp. – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – psychot. humans – acute
 admin. – chronic admin. – in vivo – blood lev. – metab. proc. – tranquilizers – *CAAAL-10238-U1
 A-0608.
- The “official method” of blood-alcohol level measurement in France is questioned by the authors on the basis of their findings with a group of 29 hospitalized patients on a regimen of tranquilizers. After a month of close observation, their blood alcohol levels were measured, and 15 had appreciable levels as indicated by the test; of these, 3 had definitely ingested alcohol and a fourth was suspect, but, for the remaining 11, no possible cause could be found for the elevated blood alcohol levels other than the tranquilizers. The authors conclude that, “the influence of certain drug complexes, and even of certain isolated drugs, on the blood alcohol level seems incontestable,” and, hence, chemical tests for drunkenness may be the cause of “regrettable judicial errors”. Their results are refuted by: Chatagnon, C., and Chatagnon, P. -A. (Ann. Medicopsychol. (Paris), 120/1: 554-559, 1962); Delay, J., Deniker, P., and Leyrie, J. (Ann. Medicopsychol. (Paris), 120/1: 752-755, 1962); and LeBreton, R., Rondepierre, J. -J., Ropert, R., and Nizard, I. (Ann. Medicopsychol. (Paris), 120/1: 755-759, 1962).
240. Chantourelle
 EXTRAIT DU RAPPORT DE M. CHANTOURELLE, SUR LE MÉMOIRE PRÉCÉDENT.
 [Extract of the statement of M. Chantourelle on the preceding report].
 Journal Général de Médecine, de Chirurgie, et de Pharmacie; ou Recueil Périodique de la Société de
 Médecine de Paris (Paris), 73: 178-183 (0 ref.), 1820.
 F – general – DC (unchanged) – humans – G.I. tract. – stimulants – *CAAAL-0 A-0609.

In reply to the report of Girard (*Journal Général de Médecine, de Chirurgie, et de Pharmacie; ou Recueil Périodique de la Société de Médecine de Paris* (Paris), 73: 166-178, 1820), the author describes some simple experiments to illustrate the action of ammonia on wine and alcohol in relation to inebriety. Ammonia added to wine changed the colour and caused a precipitation of the tartrate and carbonate salts, but did not interact with alcohol, nor destroy the aroma of wine. Thus it is pointed out that ammonia destroys the salts and acidity in the wine, but does not exercise any action on the alcohol nor the aromatic element. Moreover, the author speaks of experiences where a few drops of ammonia added to wine to strengthen the aroma caused death upon ingestion. The author explains that death was due to the liberation of appreciable quantities of hydrogen sulphide in the stomach and intestines.

241. Chapheau, M.

ACTION DE LA PHLORHIZINE ET DE L'ACIDE IODACÉTIQUE SUR LA COMBUSTION DE L'ALCOOL ÉTHYLIQUE CHEZ LE LAPIN. [Action of phlorhizin and iodoacetic acid on combustion of ethyl alcohol in rabbits].

C. R. Soc. Biol. (Paris), 116: 887-889 (3 ref.), 1934.
F – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – metab. proc. – miscellaneous
– *CAAAL-1134-A2 A-0610.

Rabbits received 2 cc of absolute alcohol/kg in 10% sol by stomach tube. Phlorhizin or iodoacetic acid was then administered. The results were negative. No modification of rate of alcohol combustion was observed after the body sugar was lowered by the action of phlorhizin, or increased by the action of iodoacetic acid.

242. Chapheau, M.

LA COMBUSTION DE L'ALCOOL ÉTHYLIQUE CHEZ LE LAPIN AU COURS DE QUELQUES INTOXICATIONS (INTOXICATION PAR LE NITRATE D'URANE ET PAR LE PHOSPHORE). [The combustion of ethyl alcohol in rabbits after some intoxications (intoxication by uranium nitrate and by phosphorus)].

C. R. Soc. Biol. (Paris), 116: 889-890 (1 ref.), 1934.
F – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – blood lev. – liver, kidney – metab. proc. – unclass. ther. agents – *CAAAL-1133-A2 A-0611.

Rabbits, wt about 2 kg, received 2 cc of absolute alcohol in 10% sol by stomach tube. After 2, 5, and 7 hr, blood alcohol content was determined. The same procedure was employed after acute intoxication with uranium nitrate, 5 to 6 mg sc. Chronic intoxication with phosphorus was produced by injection of 1 cc phosphorus oil (1:1,000) every second day. The rate of disappearance of alcohol was not appreciably modified during the acute nephritis following uranium poisoning. In phosphorus poisoning, the rate of combustion was markedly decreased.

243. Chapheau, M.

RECHERCHES EXPÉRIMENTALES SUR LA COMBUSTION DE L'ALCOOL ÉTHYLIQUE AU COURS DE QUELQUES INTOXICATIONS. [Experimental investigation of ethyl alcohol oxidation in the course of certain poisonings].

Dissertation, Faculty of Medicine and Pharmacy of the University of Bordeaux, France, 174 pp. (148 ref.), 1935.

F – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – cardiovasc. – liver, kidney – respir. – anesthetics – barbiturates – hormones, hormone antag. – unclass. ther. agents – *CAAAL-2819-A1 A-0612.

The literature is reviewed, and experiments on rabbits and guinea pigs are reported. Rabbits subjected to poisoning by 1 cc 1% phosphorous oil showed normal oxidation of alcohol until serious toxic

symptoms appeared. Administration of 2 cc/kg ethanol produced no improvement in the condition, and failed to check or retard the poisoning. Experiments on guinea pigs verified this result. Further experiments with rabbits showed that ethanol had a similar effect on chloroform poisoning. It was found that only dangerously large doses of insulin modified the oxidation of alcohol. Experimental nephritis provoked by uranium nitrate had no modifying effect on alcohol combustion. The alcohol combustion did not appreciably modify the glycemic effects of sodium iodoacetate. When light doses (0.35-0.40 cc/kg) of numal were given following 2 cc alcohol, alcohol combustion was unchanged. When the quantity of barbiturate was increased (0.63-0.83 cc/kg), alcohol oxidation was clearly diminished during deep sleep, and when respiration, circulation, and other essential functions were slowed down.

244. Chapman, L. F.

EXPERIMENTAL INDUCTION OF HANGOVER.

Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 67-86 (55 ref.), 1970.
 E – exp. comp. – congen. stud. – humans – acute admin. – in vivo – blood lev. – other drug lev. –
 mot. perform. – psychol. perform. – absorp., distrib., stor. – acid-base, blood pH, elect. – cardiovasc.
 – CNS – G.I. tract – liver, kidney – metab. proc. – nerv. syst. – respir. – senses – skel., muscle, skin
 – *CAAAL-12890-D1 B-0507.

The possible role of alcoholic congeners in the etiology of hangover was studied in 91 healthy adults. The subjects, who were occasional or moderate drinkers, drank from 1.0-1.75 ml/kg of either vodka (low congener content) or bourbon (high congener content). The beverages were consumed in a "party" atmosphere between 8 pm and 2 am, with simple behavioral tasks completed before, and 75 min after the drinking had begun. Hangover severity was assessed subjectively the next morning on a rating scale and a checklist of symptoms. No significant differences were apparent on the behavioral tests between the groups receiving vodka and bourbon. After 1.5 ml/kg of alcohol, hangover occurred in 50% of the subjects, with a greater incidence of definite hangover resulting from bourbon (20 of 30) than from vodka (13 of 30). It is concluded that a greater incidence and severity of hangover can be associated with bourbon than with vodka; however, the role of congeners in accounting for this difference remains to be determined.

245. Chappell, A. G.

SEVERE HYPOTHERMIA DUE TO COMBINATION OF PSYCHOTROPIC DRUGS AND ALCOHOL.

Brit. Med. J. (London), 1: 356 (2 ref.), 1966.
 E – general – case hist. – DC (add., infra-add., unspec. incr.) – psychot. humans – cardiovasc. – liver,
 kidney – skel., muscle, skin – antidepressants – barbiturates – tranquilizers – *CAAAL-0
 B-0262.

Reported is the case of a 43 yr-old woman with severe hypothermic coma after the combined use of alcohol and drugs. The patient had been taking sodium amytal (60 mg, 3 times/day), isocarboxazid (10 mg, 3 times/day), and perphenazine (4 mg, 3 times/day) for about 5 months. 10 days before admission, she discontinued the isocarboxazid, and 3 days later, started taking amitriptyline (25 mg, 3 times/day, increasing to 25 mg, 4 times/day). She also drank large quantities of red wine, and had no subsequent recollection of the events which led up to her losing consciousness. Admission and laboratory data are given; progress and treatment (soluble penicillin and steroids) are discussed. The patient was discharged after 15 days. The dangers of the combined use of alcohol and psychotropic drugs are stressed.

246. Chatagnon, C., and Chatagnon, M. P. -A.

A PROPOS DU PROCÈS-VERBAL DE LA DERNIÈRE SÉANCE ET DE LA
 COMMUNICATION DE MM. J. CERTHOUX ET M. RAMET. [Concerning the proceedings

of the last meeting and the report of J. Certhoux and M. Ramet].

Ann. Medicopsychol. (Paris), 120/1: 554-559 (0 ref.),

1962.

F – exp. comp. – DC (unchanged) – humans – chronic admin. – in vivo – blood lev. – metab. proc.
– autocoids – barbiturates – tranquilizers – *CAAAL-10238-U1 A-0613.

To confirm or refute the observations of Certhoux and Ramet, 11 subjects were given various drugs (barbiturates, antihistamines, tranquilizers) for 20 days. Blood alcohol concentrations showed a persistent fluctuation between 0 and 20 mg%, irrespective of the drugs taken. Nicloux's method was used for the determination. When 1 subject was given measured amounts of alcohol, blood alcohol rose as expected. The conclusions of Certhoux, J., and Ramet, M. (Ann. Medicopsychol. (Paris), 120/1: 359-365, 1962) that drugs, such as tranquilizers, make blood level values found according to the official method of determination erroneous, are thus refuted.

247. Chauchard, P., Mazoué, H., and Lecoq, R.

LES MÉDICATIONS ACCÉLÉRATRICES ET INHIBITRICES DE L'INTOXICATION ALCOOLIQUE. [Drugs as accelerators and inhibitors of alcoholic intoxication].

C. R. Soc. Biol. (Paris), 143: 1550-1553 (8 ref.),

1949.

F – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged)
– mammals – acute admin. – chronic admin. – in vivo – dose resp. – CNS – alcohols – anticonvulsants
– cardiovasc. agents – gastrointest. agents – unclass. ther. agents – *CAAAL-5355-D2 and 5415-D2
A-0614.

The effects of drugs upon the nervous perturbations caused by the ingestion of alcohol were investigated in rats. Apomorphine (0.5 cg), tetraethylthiuramdisulphide (0.5 cg), and octyl alcohol (50 g), all given sc, increased the action of alcohol (1 cc 25% alcohol, sc). Potassium rhodanate (2 cg) and phenyl-1-amino-2-propane (5 mg), given sc, tended to inhibit acute or chronic signs induced by ethanol. Their action, however, proved limited, and was very closely related to the quantities of alcohol and drugs tested. The neutralizing effect of iv injections of magnesium sulphate was not observed when the substance was given sc. According to the authors, the substances which inhibit the nervous effects of alcohol can be considered in the same category as gardenal and strychnine.

248. Chauchard, P., Mazoué, H., and Lecoq, R.

ACTION DE DIVERS NEUROLEPTIQUES ET TRANQUILLISANTS SUR LES EFFETS NERVEUX DE L'ALCOOL ÉTHYLIQUE CHEZ LE RAT. [Action of various neuroleptics and tranquilizers on the nervous effects of ethyl alcohol in the rat].

J. Physiol. (Paris), 56(3): 320-321 (1 ref.),

1964.

F – exp. comp. – DC (decrease) – DC (unspec.) – DC (sensit.) – mammals – acute admin. – in vivo
– CNS – tranquilizers – unclass. ther. agents – *CAAAL-0 A-0615.

The effects of various psychotropic drugs on the behaviour produced by sc ethyl alcohol were observed in the rat. Drugs such as clarmil, methylpentyl carbamate, and chlorpromazine, injected 1 hr before the alcohol, neutralized the characteristic alcohol effects and were strong enough to persist after sensitization to alcohol by disulfuram. All drugs tested neutralized the effects of alcohol, except procalmadiol, reserpine, methylpentynol carbamate, benactyzine, and hydroxyzine. There was no apparent parallel between the effects on alcohol and the mode of action of the drugs on the nervous centers.

249. Chelton, L. G., and Whisnant, C. L.

THE COMBINATION OF ALCOHOL AND DRUG INTOXICATION.

Southern Med. J. (Birmingham), 59(4): 393 (2 ref.),

1966.

E – stat. surv. – conj. addict. – DC (add. infra-add., unspec. incr.) – drug-dep. humans – blood lev.
– other drug lev. – barbiturates – tranquilizers – *CAAAL-0 B-0263.

100 alcoholic patients consecutively admitted to a private hospital were screened for barbiturates, meprobamate, and phenothiazine. 38% were found to have been taking one or more of these drugs, although only 9% admitted this. 27 patients had positive chromatograms for barbiturates: 19 for barbiturates alone, 5 in combination with meprobamate, 2 in combination with phenothiazine, and 1 in combination with both phenothiazine and meprobamate. A total of 14 had positive chromatograms for meprobamate—8 were positive for this drug only, and 6 for meprobamate in combination with the other drugs. Combined intoxication with alcohol and drugs must be seriously considered in the treatment of alcoholic patients, and management of the withdrawal syndrome can be complicated by simultaneous drug withdrawal.

250. Chevalier, A.

NOTE SUR L'EMPLOI DE L'ALCALI VOLATIL (AMMONIAQUE LIQUIDE) CONTRE L'IVRESSE. [Note on the use of volatile alkali (liquid ammonia) against intoxication].

Revue Médicale Française et Étrangère (Paris), 12: 290-304 (3 ref.),

1823.

F – general – case hist. – DC (antidotal) – DC (unchanged) – humans – G.I. tract – CNS – miscellaneous – *CAAAL-0 A-0616.

The author refers to the article by Girard (Journal Générale de Médecine, de Chirurgie, et de Pharmacie; ou, Recueil Périodique de la Société de Médecine de Paris (Paris), 73: 166-178, 1820), which recommends the use of ammonia (7-8 drops in a glass of water) to counteract intoxication. Citing a number of case histories in which ammonia (6-12 drops in a glass of water) was employed in alcoholic intoxication, he believes that this cannot be regarded as a certain remedy, inasmuch as the ammonia had no effect on some subjects, whereas it did affect others. The author refers to data in the literature confirming such findings, and concludes that ammonia may be viewed as a non-specific remedy against inebriety, and that its efficacy depends upon a number of factors (e.g., susceptibility of the individual, nature and quantity of the ingested liquor, absorption, etc.).

251. Chew, W. B., Berger, E. H., Brines, O. A., and Capron, M. J.

ALKALI TREATMENT OF METHYL ALCOHOL POISONING.

J. A. M. A. (Chicago), 130(2): 61-64 (9 ref.),

1946.

E – general – case hist. – DC (antidotal) – post-mort. – humans – blood lev. – other drug lev. – blood comp., sites, lymph. – cardiovasc. – CNS – G.I. tract – liver, kidney – metab. proc. – respir. – senses – skel., muscle, skin – alcohols – elect., water-bal. agents – *CAAAL-4316-E4 A-1245.

The clinical, pathological, and therapeutic data concerning 31 patients with methanol poisoning are presented. 5 died from respiratory failure within 3 hr of hospital admission, and the rest recovered. Methanol consumption varied from 90-540 cc. Treatment commenced 11-37 hr after the first ingestion of methanol. No correlation existed between the amount of methanol ingested and the blood CO₂-combining power (which decreased in every case but 1), the clinical picture, or eye effects. Primary treatment was aimed at overcoming the acidosis—4 g sodium bicarbonate po or by gavage (every 15 min), and/or 1/6 M sodium r-lactate iv. A rough correlation existed between original CO₂-combining power of blood and the amount of alkali (average total 73.8 g) given on the first day. Whiskey (1 oz every 4 hr for a day or two) was administered to promote intracellular displacement of methanol. Symptoms abated within a few hr, plasma bicarbonate rose progressively, and, after 14 hr, CO₂-combining power exceeded 35 vol %. Such excellent results emphasize the importance of prompt acidosis elimination by alkali administration. There was no correlation between the relative amounts of beer and whiskey ingested and the clinical condition of the patients, or their response to treatment.

252. Chiffot, M. J.

SUR UN CAS DE RUBÉFACTION DE LA FACE TENDANT À SE GÉNÉRALISER, À LA SUITE DE L'INGESTION DU *COPRINUS ATRAMENTARIUS* FR. [A case of facial

rubor, tending to spread, following ingestion of *Coprinus atramentarius* Fr].

Société Mycologique de France, Bulletin (Paris), 32: 63 (O ref.),

1916.

F – general – DC (sensit.) – humans – cardiovasc. – *CAAAL-0

A-1271.

The mushroom *Coprinus atramentarius* is considered edible if young. Taken with an alcoholic beverage, however, it will produce intense facial rubor, extending to the neck and body if sufficient alcohol is drunk. The reddening is not accompanied by urticaria, and disappears after a few hr, but may reappear up to 48 hr without further ingestion of *Coprinus*, if wine is drunk. A gardener who ate only very small quantities of *Coprinus* did not experience any symptoms, as no alcoholic beverage was consumed at the time. The author can provide no explanation for the phenomenon.

253. Child, G. P.

THE INABILITY OF COPRINI TO SENSITIZE MAN TO ETHYL ALCOHOL.

Mycologia (New York), 44: 200-202 (9 ref.),

1952.

E – exp. – DC (unchanged) – humans – acute admin. – in vivo – cardiovasc. – respir. – unclass. ther. agents – *CAAAL-0

A-1246.

It being alleged that *Coprinus atramentarius* and *C. comatus* produce adverse reactions when alcohol is drunk after ingestion of the fungi, *C. atramentarius*, *C. comatus*, and *C. micaceus* were collected, washed, and prepared in various ways to be consumed with alcohol, in order to determine any adverse side effects in humans. The individual preparations were eaten in 100-600 g amounts by the same subject, who had previously shown his ability to become sensitized to alcohol with other drugs, his reactions being well known. Each species was tested more than 5 times with alcohol, and no adverse side effects were detected. The subject, however, also ingested 2 g parboiled *Paneolus companulatus* in a separate test, which, without alcohol, produced a flushing of the face, increased pulse rate, and induced slight dyspnea and a glassy-eyed appearance. Effects subsided in 1 hr with no sequelae. It is concluded that the sensitization reaction hitherto attributed to *Coprinus*-alcohol may be due to the accidental inclusion of *Paneolus* with *Coprinus*—an easy enough mistake, since these fungi can be similar in appearance. It is possible, however, that *Coprinus* from other regions than that from which the samples were taken could produce a reaction to alcohol.

254. Chilian, O.

ÜBER DIE BEEINFLUSSUNG DER VERGIFTUNGEN MIT NITROBENZOL, DINITROBENZOL, PARANITROCHLORBENZOL UND DINITROCHLORBENZOL DURCH ALKOHOL. [The influence of alcohol on poisoning with nitrobenzol, dinitrobenzol, paranitrochlorbenzol, and dinitrochlorbenzol].

Dissertation, Medical Faculty of the University of Würzburg, Germany, 55 p. (2 ref.),

1902.

G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – absorp., distrib., stor. – CNS – diagnost. agents – miscellaneous – *CAAAL-0

A-0617.

The four above-stated compounds were given in various doses to rabbits (by stomach tube), alone and followed by alcohol (one or several doses of 3.5 cc absolute alcohol). Alcohol given after nitrobenzol had no significant effect. Alcohol given after dinitrobenzol, paranitrochlorbenzol, or dinitrochlorbenzol, however, caused incoordination, convulsions, nystagmus, intoxication, and death. The author explains the difference in the alcohol action as being due to the fact that dinitrobenzol, paranitrochlorbenzol, and dinitrochlorbenzol need a solvent in order to be absorbed by the tissues (to have a poisonous effect), and alcohol is such a solvent. Nitrobenzol, however, is readily absorbed (without the help of a solvent), and is therefore little affected by alcohol.

255. Christenson, P. J., and Tracy, C. H.

FURALTADONE—A NEW SYSTEMIC ANTIBACTERIAL AGENT: STATISTICAL REVIEW OF 466 CASES.

Curr. Ther. Res. (New York), 2(1): 22-29 (8 ref.), 1960.
 E – SEC – stat. surv. – DC (sensit.) – humans – metab. proc. – respir. – skel., muscle, skin –
 anti-infectants – *CAAAL-0 A-1272.

An unusual reaction with alcohol is reported as a side-effect of the systemic antibacterial agent furaltadone (altafur). A detailed statistical study was made of 466 patients, aged 11 days to 94 yr, who were treated in various parts of the United States with furaltadone for a variety of bacterial infections. The drug had been administered po in divided dosages ranging from 200-2400 mg/day. 20 ambulatory patients, who had taken an alcoholic beverage while on furaltadone therapy, experienced a reaction characterized by urticaria, dyspnea, and a feeling of generalized weakness or of chest constriction. The authors interpret the reaction as an apparent interference by furaltadone with the oxidation of alcohol. This interpretation would account for the reaction resembling that produced by the sulfonylureas, such as chlorpropamide, and by disulfiram. It is therefore recommended that patients should refrain from ingesting alcohol in any form while being treated with furaltadone, and for 7 days thereafter.

256. Claeys
 EFFETS REMARQUABLES DE L'AMMONIAQUE DANS UN CAS D'IVRESSE AVEC
 DÉLIRE FURIEUX. [Remarkable effects of ammonia in a case of intoxication with wild
 delirium].
 Bulletin Général de Thérapeutique Médicale, Chirurgicale, Obstétricale et Pharmaceutique (Paris),
 44: 546-547 (0 ref.), 1854.
 F – general – case hist. – DC (antidotal) – humans – stimulants – *CAAAL-0 A-0618.

Case material is given concerning an acutely-intoxicated soldier for whom the author prescribed 20 drops of ammonia in 4 oz of water. After 3 spoonful, the patient gradually calmed down. When delirium recurred, the patient was treated with another 2 spoonful of the same potion, whereupon his condition improved at once. 4 hr later, he ate dinner as usual. The author indicates that ammonia in the given dosage is best administered po, with simultaneous inhalation if desired. Inhalation is advocated in comatose cases.

257. Clark, B. B., Morrissey, R. W., Fazekas, J. F., and Welch, C. S.
 THE ROLE OF INSULIN AND THE LIVER IN ALCOHOL METABOLISM.
 Quart. J. Stud. Alcohol (New Haven), 1: 663-683 (73 ref.), 1940.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute
 admin. – chronic admin. – in vivo – in vitro – blood lev. – liver, kidney – metab. proc. – anesthetics
 – anti-infectants – elect., water-bal. agents – hormones, hormone antag. – *CAAAL-0 A-0619.

Alcohol metabolism was investigated in dogs and cats. Injection of insulin, insulin with glucose, or insulin with glucose and sodium bicarbonate definitely increased the rate of alcohol oxidation during the subsequent 2 hr. 1 dog was given 20 cc carbon tetrachloride/day for 23 days. The rate of alcohol oxidation decreased progressively from the control value of 19.0 mg%/hr to 8.0 mg%/hr. When 1 dog was subjected to 6 periods of chloroform anesthesia for 1 1/2 hr each during a 23-day period, the rate of alcohol oxidation decreased to 9.0 mg%/hr. The fatty liver produced in 6 dogs by fasting, plus 2 g phloridzin/day for 1 week, only effected a reduction of alcohol oxidation to 13.5 mg%/hr.

258. Clark, W. C., Blackman, H. J., and Preston, J. E.
 CERTAIN FACTORS IN AGGREGATED MICE D-AMPHETAMINE TOXICITY.
 Arch. Int. Pharmacodyn. (Gand), 170(2): 350-363 (9 ref.), 1967.
 E – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – CNS
 – metab. proc. – amphetamines – *CAAAL-0 B-0264.

Aggregated mice treated with d-amphetamine (10 or 15 mg ip in saline sol) showed exaggerated excitement, more intense hyperactivity, hyperthermia, hypoglycemia, and higher mortality than segregated mice. The administration of either dexamethasone (40 g/kg), ethanol, glucose (10.0 g/kg), 2,deoxy-d-glucose (2 g/kg), or diphenylhydantoin (15.0 mg/kg) reduced the excitement, hyperactivity, and mortality in aggregated mice given d-amphetamine. Small doses of alcohol (0.5 g/kg) evoked combativeness, and larger doses (1.0 g to 2.0 g/kg) antagonized the amphetamine stimulation.

259. Cléménçon, H.

ANTABUS-WIRKUNG BEI KÜHEN? [Antabuse effect in cows?].

Schweizerische Zeitschrift für Pilzkunde (Burgdorf), 40(11): 170-172 (0 ref.),

1962.

G – general – DC (sensit.) – humans – mammals – cardiovasc. – nerv. syst. – respir. – senses – skel., muscle, skin – unclass. ther. agents – *CAAAL-0 A-1332.

The effects of antabuse (tetraethylthiuramdisulfide) and alcohol on humans are described: reddening of the skin, rapid pulse, forced breathing, rasping cough, feeling hot, headaches, and nausea. The more alcohol consumed, the harsher the effects; the amount of antabuse does not much affect the severity of the symptoms. As little as 5 g alcohol will trigger a reaction, and large quantities can be lethal. A reaction to alcohol will occur even after a few days, and this is presumably because antabuse is stored in the body for a time. A probable reason for the action of antabuse is that alcohol is oxidized in 2 steps by the body—first to acetaldehyde through the enzyme alcoholdehydrogenase, and then to acetic acid through the enzyme acetaldehydoxydase. Antabuse forms an inactive complex with acetaldehydoxydase, thus allowing the concentration of acetaldehyde to build up in the organism. Acetaldehyde is poisonous, and its toxic effects are the same as those previously described. The illness of a cow that had eaten *Coprinus atramentarius* mushrooms and then was treated with brandy is described as being similar to the effects of antabuse and alcohol, probably due to the presence of antabuse in *Coprinus*.

260. Clemm, W. N.

WEINGEIST ALS SCHUTZMITTEL GEGEN GIFTIGE EIWEISSKÖRPER. [Spirit of wine as a medicinal prophylactic against toxic albumin bodies].

Pfluegers Archiv für die Gesamte Physiologie des Menschen und der Tiere (Bonn), 93: 295-301 (11 ref.),

1902.

G – general – DC (antidotal) – humans – metab. proc. – *CAAAL-0 A-0620.

This paper is a commentary on the effectiveness of alcohol against albuminous toxins, (e.g. snake bites, stings) and auto-intoxication by poisonous toxins. That wine, rum, whiskey, etc., are highly effective antidotes is proven by a large body of evidence drawn from the literature. The author concludes with an elucidation of the reaction mechanism whereby alcohol precipitates out albuminous toxins from the organism.

261. Coldwell, B. B., Trenholm, H. L., and Wiberg, G. S.

THE EFFECT OF ETHANOL ON THE TOXICITY OF BARBITURATES.

Fifth International Meeting of Forensic Sciences, Toronto, Ontario, Canada, 13 pp. + 12 pp. of graphs and tables (22 ref.),

1969.

E – exp. cont. – exp. comp. – presentation – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – dose resp. – blood lev. – other drug lev. – CNS – metab. proc. – barbiturates – sed., hypnot. – *CAAAL-0 B-0265.

A systematic investigation, presently underway, of the interaction of ethanol with 5 commonly-prescribed barbiturates in rats is discussed. Initial studies indicate that ethanol exerts its greatest effect on the pharmacological activity of long-acting poorly-metabolized barbiturates. Thus, ethanol produced a dose-related decrease in the LD₅₀, a shortening of the induction time, and a prolongation

of the sleeping time of thiopental, pentobarbital, amobarbital, phenobarbital, and barbital, the response being most pronounced with the latter 2 compounds. Barbiturate levels in the brain were elevated significantly in the presence of ethanol. It is concluded that the foregoing interactions are manifested at the CNS level.

262. Coldwell, B. B., Genest, K., and Hughes, D. W.
EFFECT OF *COPRINUS ATRAMENTARIUS* ON THE METABOLISM OF ETHANOL IN MICE.
J. Pharm. Pharmacol. (London), 21(3): 176-179 (7 ref.), 1969.
E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (sensit.) – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – CNS – G.I. tract – metab. proc. – unclass. ther. agents – *CAAAL-0 B-0570.

The toxic and ethanol-potentiating effects of the fungus *Coprinus atramentarius* (inky cap) were studied in non-fasted male albino mice. Freshly-prepared samples of the dried, powdered fungus (IC/A), the ethanol-insoluble fraction (IC/B), and the ethanol-soluble fraction (IC/C) were suspended (1 g/10 ml) in 0.25% aqueous gum tragacanth, and fed to different groups of mice (2.5 ml/30 g body wt). Ethanol (30% w/v, 6 g/kg body wt) was administered po 4 hr later. Measurements of the incidence of sleeping and the sleeping time were found to be greatest in the IC/C plus ethanol group, followed by IC/A plus ethanol, IC/B plus ethanol, and ethanol alone. Administration of IC/C (3.5 g/kg) resulted in a considerable increase in the blood acetaldehyde concentration, beginning 15 min after ethanol (6 g/kg) administration, and in an elevated blood ethanol concentration at 4-8 hr. It is concluded that the ethanol-soluble fraction of *C. atramentarius* potentiates the action of ethanol in mice, possibly by decreasing the rate of absorption of ethanol, and inhibiting the oxidation of acetaldehyde.

263. Coldwell, B. B., and Platonow, N.
THE EFFECT OF METHYLMERCURIC ACETATE ON THE RATE OF DISAPPEARANCE OF ETHANOL FROM THE BLOOD OF SWINE.
Toxic. Appl. Pharmacol. (New York), 14(2): 368-375 (28 ref.), 1969.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – *CAAAL-0 B-0509.

Male piglets (13.7 ± 416 kg) were given ethanol (1 g/kg ip) and methylmercuric-203 acetate (MMA; equivalent to 5 mg mercury/kg ic), separately and in combination. Periodic analyses were made for blood ethanol (BEC) and acetaldehyde concentrations by cardiac puncture, and the animals were killed after 6 hr. It was found that the distribution of mercury in the tissues and the rate of disappearance of mercury from the blood were not affected by ethanol. After administration of ethanol alone, the BEC reached a peak in 30 min, remained constant for 30 min, then decreased at the rate of 16 ± 6 mg%/hr. In the presence of MMA, the BEC also reached a peak in 30 min, but was 2.4 times higher than in controls; the BEC dropped sharply in the next 15 min, and thereafter disappeared at the rate of 23 ± 1 mg%/hr. It is concluded that the elevated blood ethanol levels after the combined dose probably did not result from a reduced ethanol metabolism or a reduced excretion of non-metabolized ethanol, but either from a more rapid ethanol absorption into the blood, or from an alteration in the body distribution of ethanol, induced by MMA. Acetaldehyde was present in the blood under all drug conditions.

264. Coldwell, B. B., Wiberg, G. S., and Trenholm, H. L.
SOME EFFECTS OF ETHANOL ON THE TOXICITY AND DISTRIBUTION OF BARBITURATES IN RATS.
Canad. J. Physiol. Pharmacol. (Ottawa), 48(4): 254-264 (38 ref.), 1970.
E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – CNS – metab. proc. – anesthetics – sed., hypnot. – *CAAAL-0 B-0508.

In 1 of 3 experiments, rats received ip: amobarbital (A—60 mg/kg), barbital (B—125 mg/kg, pentobarbital (Pt—30 mg/kg), phenobarbital (Ph—50 mg/kg), or thiopental (T—30 mg/kg); other rat groups received the above barbiturates plus 3 g/kg ethanol (E) ip as a 15% w/v sol. Loss of righting reflex (LRR), regain of righting reflex (RRR), induction times (IT) and sleeping times (ST) were determined. In another experiment, rats received: E, Ph, Ph plus E, or no treatment, using above dosages, and blood and brain decay profiles were analyzed. In a third test using above doses, rats received: E, Pt, Pt plus E, T, T plus E, or no treatment; blood and brain levels after RRR were determined. It was found that IT was shortened and ST lengthened after all ethanol-barbiturate combinations, the greatest effect being with E-B and E-Ph. Brain levels of B, Ph, acetaldehyde, and acetone were increased by E. Rats given A, Pt, or T had significantly higher brain and serum barbiturate levels at the time of RRR than rats receiving E plus A, Pt, or T, indicating that CNS depression is not only dependent on brain barbiturate concentration. Decay profiles of serum barbiturate levels were not altered by E, and barbiturates had no effect on E brain or blood levels. Ph-E depressed blood and brain acetaldehyde, but not acetone levels. Pt, T, Pt-E, and T-E increased brain, but not blood acetaldehyde. Ph failed to affect blood acetone or acetaldehyde, but increased brain levels; Ph-E decreased blood acetone. T increased blood acetone, and T-E decreased brain acetone.

265. Cole, L. J., and Ellis, M. E.

ADDITIVITY OF RADIATION PROTECTION BY CYSTEINE AND SODIUM NITRITE IN MICE.

Amer. J. Physiol. (Bethesda), 175: 429-436 (20 ref.),

1953.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – blood comp., sites, lymph – metab. proc. – respir. – cardiovasc. agents – *CAAAL-6971-D2 A-0621.

X-irradiated mice were pretreated with cysteine (900 mg/kg iv) and sodium nitrite (100 mg/kg ip), alone or in combination; and with ethanol (3.0 ml in 25% dilution in 0.9% saline/100 g ip, given 30 min before irradiation), alone or in combination with sodium nitrite. The cysteine-sodium nitrite combination was found to afford an additive protection against mortality at supralethal radiation dose levels at which neither substance alone gives protection. In contrast, at the supralethal dose level (1000 roentgens), no significant decrease in mortality was observed among the mice pretreated with sodium nitrite plus ethanol, as compared with those receiving either substance alone. Furthermore, no significant difference in the average survival time, or in body wt changes were observed when either or both of the two compounds were administered.

266. Comporti, M., Hartman, A., and Di Luzio, N. R.

EFFECT OF IN VIVO AND IN VITRO ETHANOL ADMINISTRATION OF LIVER LIPID PEROXIDATION.

Lab. Invest. (New York), 16: 616-624 (50 ref.),

1967.

E – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – in vitro – liver, kidney – metab. proc. – *CAAAL-0 B-0266.

Liver lipid peroxidation was estimated by the amount of malonaldehyde formed by incubated liver homogenates from 80 fasting female rats which had received by intubation either 6 g of 50% ethanol/kg or isocaloric glucose. In 20 in vitro studies conducted in duplicate, addition of 50 μ l of ethanol increased thiobarbituric acid to 135% of control values after 1 hr. Ethanol and N,N-diphenyl-p-phenylenediamine (DPPD) combined reduced the level to 17.6% of control values. Liver triglyceride concentration, 188% higher in the ethanol than in the glucose group, was maintained at normal levels in the ethanol-DPPD rats. The studies indicate that lipoperoxidation is possibly a fundamental mechanism in acute ethanol-induced fatty liver.

267. Cooke, A. R.
ASPIRIN, ETHANOL AND THE STOMACH.
 Aust. Ann. Med. (Sydney), 3: 269-274 (55 ref.), 1970.
 E – SEC – review – DC (add., infra-add., unspec. incr.) – mammals – G.I. tract – metab. proc. – analg., antipyret. – *CAAAL-0 B-0918.

The data on the separate metabolism and effects of ethanol and aspirin—absorption by the stomach, gastric emptying, effect on gastric secretion, and effect on gastric disease—are reviewed. Despite the widespread use of both drugs, few studies have examined their combined effects. Goulston and Cooke (Brit. Med. J., 4(5632): 664-665, 1968) found that, although ethanol by itself did not increase faecal blood loss, it significantly increased faecal blood loss caused by unbuffered aspirin. Davenport (Gastroenterology, 56(3): 439-449, 1969) investigated the combined effect of aspirin and ethanol in dogs with Heidenhain pouches, and found that ethanol significantly increased aspirin-induced bleeding. Whether these findings are of clinical importance in regard to overt bleeding has yet to be determined. It is concluded that both ethanol and aspirin “break” the gastric mucosal barrier to hydrogen ion diffusion from lumen to blood, and that both can cause acute gastritis. Ethanol alone does not cause occult bleeding, whereas unbuffered aspirin does cause occult bleeding. Also, ethanol probably does not cause chronic gastritis or peptic ulcer, whereas aspirin may be associated with chronic gastritis ulcer. Ethanol accentuates occult blood loss and damage to gastric mucosa induced by unbuffered aspirin.

268. Cooper, A. J., Magnus, R. V., and Rose, M. J.
A HYPERTENSIVE SYNDROME WITH TRANLYCYPROMINE MEDICATION.
 Lancet (London), 1(7332): 527-529 (10 ref.), 1964.
 E – SEC – general – DC (add., infra-add., unspec. incr.) – humans – cardiovasc. – enzymes – *CAAAL-0 A-0622.

Of 137 out-patients treated with tranlycypromine, 20% developed severe toxic symptoms suggesting hypertensive crises. The case histories of these 27 patients are tabulated. 9 additional patients were seen and examined during an actual attack. One of these, a 45 yr-old male, had an attack precipitated by alcohol ingestion 56 hr previously; he had occipital headache and grand mal, complicated by subarachnoid hemorrhage. His attack lasted 7 days, and he was completely recovered in 3 weeks.

269. Cooper, J. R., and Kini, M. M.
BIOCHEMICAL ASPECTS OF METHANOL POISONING.
 Biochem. Pharmacol. (New York), 11: 405-416 (69 ref.), 1962.
 E – SEC – review – DC (antidotal) – humans – mammals – species or sex diff. – absorp., distrib., stor. – acid-base, blood pH, elect. – CNS – metab. proc. – alcohols – *CAAAL-0 A-1382.

Present knowledge of the biochemical aspects of methanol intoxication—absorption and excretion, metabolism, clinical characteristics of poisoning, pathology, treatment, formaldehyde as the toxic agent, species difference, metabolic lesion in blindness, and metabolic acidosis—is reviewed and discussed. Laboratory investigations by the authors have led to the conclusion that it is alcohol dehydrogenase, and not the catalase system, that is responsible for the physiological oxidation of methanol. The use of ethanol as an adjunct to bicarbonate treatment in methanol toxicity is mentioned briefly. It is noted that, although ethanol delays methanol oxidation, thereby increasing the excretion of the latter in rabbits, monkeys, and humans, it should be remembered that administration of ethanol would also enhance the degree of depression of the CNS in an already comatose patient, resulting in a possibly fatal outcome.

270. Copeman, P. R. v. d. R.
POISONING BY BARBITURATES.
 J. Forensic Med. (Johannesburg), 1(5): 271-283 (6 ref.), 1954.

E – stat. surv. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – post-mort. – humans – other drug lev. – absorp., distrib., stor. – G.I. tract – liver, kidney – barbiturates – *CAAAL-0

A-0623.

From 1948 to 1952, 112 cases of suicide or death through overdosage caused by barbiturates were examined at the Government Chemical Laboratories in Johannesburg. The results of 109 cases are presented and discussed; of these, 18 cases showed the presence of alcohol. In the alcohol group, the concentration of barbiturates in the kidneys tended to be slightly higher than in the liver (23.2 parts/million); the average concentration in the stomach was relatively high, and concentrations in the liver and kidneys appeared to be slightly lower (28.8 and 29.9 parts/million)—these differences are shown, however, to be of no significance. It is concluded that the chemical results, considered by themselves, do not indicate any significant differences due to the presence of alcohol; under the circumstances, there was no possibility of control, and variations in statistical groups were due to these random factors. Thus, no general conclusions can be drawn regarding alcohol-barbiturate combinations.

271. Corkill, N. L.

SNAKE BITE IN THE TROPICS.

Practitioner (London), 183: 354-358 (1 ref.),

1959.

E – SEC – general – DC (add., infra-add., unspec. incr.) – humans – metab. proc. – stimulants –

*CAAAL-0

A-0624.

A description of the various families of poisonous snakes, and of treatment for poisonous snake bites, is given. Treatment is based on the assumption that depression of muscular and biochemical activity is conducive to limitation of the speed of fixation of venom, and thus more time is given for systematic adjustment and the arrival of antivenom. The author regards the use of alcohol and stimulants as harmful because they increase metabolic activity.

272. Cornish, H. H., and Adefuin, J.

ETHANOL POTENTIATION OF HALOGENATED ALIPHATIC SOLVENT TOXICITY.

Amer. Industr. Hyg. Ass. J. (Detroit), 27: 57-61 (10 ref.),

1966.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – blood comp., sites, lymph – liver, kidney – metab. proc. – respir. – analg., antipyret. – anti-infectants – *CAAAL-0

B-0267.

Male rats were used to investigate the potentiating action of prior ethanol ingestion on the toxicity of carbon tetrachloride, trichloroethylene, perchloroethylene, and 1,1,1-trichloroethane. Ethanol potentiation of toxicity, as measured by serum enzyme response, could be demonstrated if rats were exposed 18 hr later to carbon tetrachloride or trichloroethylene, but not if exposed to perchloroethylene or 1,1,1-trichloroethane. Subsequent exposure of ethanol-treated rats for 8 hr to 25 or 50 parts/million of carbon tetrachloride or to 100 parts/million of trichloroethylene failed to affect serum levels. However, ethanol potentiation of carbon tetrachloride toxicity could be demonstrated after as slight an exposure as 2 hr at 100 parts/million, and potentiation of trichloroethylene toxicity was apparent after a 4-hr exposure to 5000 parts/million.

273. Cornish, H. H., and Adefuin, J.

POTENTIATION OF CARBON TETRACHLORIDE TOXICITY BY ALIPHATIC ALCOHOLS.

Arch. Environ. Health (Chicago), 14: 447-449 (5 ref.),

1967.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – blood comp., sites, lymph – metab. proc. – anti-infectants –

*CAAAL-12295-B2

B-0268.

Ethanol; methanol; normal propyl and isopropyl alcohol; normal, iso-, secondary, and tertiary butyl alcohol; and amyl alcohol were administered po at relatively high dosage levels (40% of the LD₅₀) to rats which were then exposed to 1000 parts/million carbon tetrachloride (CCl₄ vapour. The serum glutamic oxaloacetic transaminase (SGOT) was used as a measure of tissue response. A 6-rat group exposed to CCl₄ vapour for 2 hr, 16-18 hr after ingestion of 5.0 g/kg ethanol, showed a SGOT level of $1,941 \pm 558$. Another group exposed to CCl₄ 2 hr after ethanol ingestion (5.0 g/kg) showed a SGOT level of 202 ± 26 . Thus, potentiation was marked when ethanol ingestion took place 16-18 hr prior to CCl₄, but was not evident when ethanol was given 2 hr before CCl₄. Similar results were demonstrable with the other alcohols tested.

274. Cossa, P., Bougeant, H., Puech, M., and Sassi, P.
LE TRAITEMENT DES COMPLICATIONS NERVEUSES DE L'ALCOOLISME PAR LA STRYCHNINE. [Treatment of nervous complications of alcoholism by strychnine].
Ann. Medicopsychol. (Paris), 96/1: 167-187 (19 ref.), 1938.
F – general – case hist. – DC (decrease) – drug-dep. humans – dose resp. – psychol. perform. – blood comp., sites, lymph – CNS – liver, kidney – metab. proc. – stimulants – *CAAAL-878-N7
A-0625.

Case reports are presented of 15 alcoholics who received injections of strychnine sulfate (0.03 to 0.05 g daily) for acute alcoholism (delirium tremens), and chronic alcoholism. The treatments were considered successful. It is suggested that strychnine forms a chemical compound with alcohol and acts like a chemical antidote. There is functional antagonism between alcohol and strychnine; alcohol increases the chronaxy, but strychnine decreases it.

275. Courvoisier, S., Fournel, J., Ducrot, R., Kolsky, M., and Koetschet, P.
PROPRIÉTÉS PHARMACODYNAMIQUES DU CHLORHYDRATE DE CHLORO-3-(DIMÉTHYLAMINO-3^o PROPYL)-10 PHÉNOTHIAZINE (4,560 R.P.): ÉTUDE EXPÉRIMENTALE D'UN NOUVEAU CORPS UTILISÉ DANS L'ANESTHÉSIE POTENTIALISÉE ET DANS L'HIBERNATION ARTIFICIELLE. [Pharmacodynamic properties of 3-chloro-10-(3-dimethylaminopropyl)-phenothiazine hydrochloride (4,560 R. P.): experimental study of a new substance used in potentiated anesthesia and in artificial hibernation].
Arch. Int. Pharmacodyn. (Gand), 92(3-4): 305-361 (80 ref.), 1953.
F – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – tranquilizers – *CAAAL-0
A-0626.

Studies were made of the changes which occur at the threshold of intoxication induced by appreciable doses of alcohol, and in acute alcohol intoxication after administration of chlorpromazine. Rats were given 5 ml alcohol sol (2-30% concentration)/100 g body wt 15 min prior to sc injection with 10 mg/kg chlorpromazine. The chlorpromazine potentiated alcohol effects by altering the intoxication symptoms through suppression of the psychomotor excitation phase, thus producing a very rapid stupefaction. It appreciably extended the duration of narcosis and increased the alcohol toxicity.

276. Coutinho, E. M., Filho, J. A., and Xavier, R.
EFFECT OF ETHANOL ON THE RESPONSE OF THE NON-PREGNANT HUMAN UTERUS TO OXYTOCIN AND VASOPRESSIN.
J. Obstet. Gynaec. Brit. Comm. (London), 77(2): 164-166 (6 ref.), 1970.
E – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – acute admin. – in vivo – mot. perform. – glands – elect., water-bal. agents – stimulants – *CAAAL-0
B-0919.

Studies were conducted during the menstrual cycle of 11 normal non-pregnant women to determine the effect of alcohol on uterine activity influenced by neurohypophysial hormones (oxytocin and

vasopressin). After spontaneous uterine activity was recorded for 2-3 hr, the response to various doses of the hormones was tested. Alcohol sol (75 g 95% alcohol/l) was infused iv at a rate of 0.55-0.60 g alcohol/kg/hr for 2 hr, resulting in a blood alcohol concentration (BAC) of 0.1-1.7 mg/ml. When spontaneous uterine activity had disappeared, the threshold doses of oxytocin and vasopressin were administered again. In 5 of the 11 subjects, the response to the hormones was unchanged by alcohol. In 4 instances, alcohol enhanced uterine response. This potentiation was seen more often with oxytocin than with vasopressin, and may have been due to the fact that the uterus was quiescent after alcohol administration, and, therefore, the response was stronger than the response superimposed on a strong spontaneous activity. In 2 instances, a diminution was observed, but this was no more than that observed without alcohol, and was probably a tachyphylactic phenomenon. It is concluded that the BAC of alcohol used therapeutically in threatened premature labour (1.0-1.8 mg/ml) does not reduce the reactivity of the non-pregnant human uterus, and that the suppression of uterine activity by alcohol is mediated by neurohypophysial inhibition.

277. Craig, R. D.

PAGITANE IN THE TREATMENT OF ALCOHOLISM: A PRELIMINARY REPORT.

Quart. J. Stud. Alcohol (New Haven), 17: 24-27 (4 ref.),

1956.

E – general – case hist. – DC (sensit.) – humans – drug-dep. humans – nerv. syst. – senses – antispasmodics – *CAAAL-7223-M3 A-1383.

6 case summaries are presented in which the toxic side-effects of pagitane hydrochloride were potentiated by alcohol. Of the 6 patients, 1 had Parkinson's disease (paralysis agitans), 1 had Thomsen's disease (myotonia congenita), and 4 were alcoholics. The effects induced by alcohol included: ataxia, dizziness, disorientation, mild nausea, and vomiting. In 2 cases, the taste of alcohol became offensive—to the patient with Thomsen's disease, alcohol tasted like kerosene, and he was not able to consume alcohol until the pagitane had been discontinued for 72 hr; the other such patient, an alcoholic, found that, when he attempted to drink beer (from the can, as usual), the previously pleasant taste was substituted by a taste like tin metal. A review of the literature of the past 3 years does not reveal any study on the use of pagitane for the treatment of alcoholics, and, on the basis of these cases, it is suggested that the drug may be a valuable and safe adjunct in treatment. Caution is urged in individualizing the dosage to limit toxic side-effects. Since pagitane inhibits the parasympathetic nervous system, it should be avoided in cases of incipient glaucoma, urinary retention, and tachycardia.

278. Cramer, E.

DIE WIRKUNG DES ALKOHOLS BEI DER SCHLAFMITTELVERGIFTUNG. [The effect of alcohol in sedative poisoning].

Dissertation, Medical Faculty of the University of Göttingen, Germany, 18 pp. (18 ref.), 1938.

G – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – CNS – barbiturates – sed., hypnot. – *CAAAL-0 A-0627.

The effect of small doses of alcohol on three sedatives was tested in rats and rabbits. Rats which had received single (250 mg/kg) or double lethal doses of veronal sodium were kept alive by alcohol in doses of 1.0 or 2.0 cc of various concentrations. Alcohol had the strongest effect in shortening the sleeping time of chloral hydrate when the latter was given in the lowest dosage (200 mg/kg). Alcohol also shortened the sleeping time induced by evipan in rabbits. In general, lower alcohol concentrations were more effective than higher ones.

279. Creaven, P. J., and Roach, M. K.

THE EFFECT OF CHLORAL HYDRATE ON THE METABOLISM OF ETHANOL IN MICE.

J. Pharm. Pharmacol. (London), 21: 332-333 (6 ref.),

1969.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – sed., hypnot. – *CAAAL-14443 B-0510.

30 min after receiving 200, 400, or 600 mg/kg chloral hydrate ip, adult 25 g male mice were injected with 1.33 g/kg ethanol iv. Blood samples were taken at 5, 20, 35, and 50 min, and ethanol and acetaldehyde concentrations were determined. Increases in blood acetaldehyde levels of 131%, 60%, 42% and 27% were seen at 5, 20, 35, and 50 min, respectively, with the 200 mg/kg chloral hydrate dose. With the 400 mg/kg dose, increases for the same time intervals were 318%, 341%, 121%, and 55%, respectively; with the 600 mg/kg dose, they were 270%, 342%, 171% and 93%. When 200 mg/kg chloral hydrate and twice the ethanol dose were given, the acetaldehyde level was greater than control values only at the 5 min interval, while the 5, 20, 35, and 50 min acetaldehyde levels were lower than those of the 200 mg/kg group with the 1.33 g/kg ethanol dosage. The 200, 400, and 600 mg/kg chloral hydrate doses increased the blood ethanol level 10, 16, and 22%, respectively, at 5 min. The rate of decrease in blood ethanol was constant and the same as for controls with the 200 mg/kg dose, whereas, with the 400 and 600 doses, the rate of decrease was not constant, being greatest for the first 15 min and declining thereafter.

280. Creaven, P. J., and Barbee, T.
THE EFFECT OF ETHANOL ON THE METABOLISM OF AMPHETAMINE BY THE RAT.

J. Pharm. Pharmacol. (London), 21(12): 859-860 (5 ref.), 1969.
E – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – other drug lev. – metab. proc. – amphetamines – *CAAAL-14591 B-0496.

In experiments using 150 g male rats, the test group was pretreated with 5 g/kg of 25% ethanol via stomach tube, the control group with isotonic saline. 30 min later, both groups were given C¹⁴-labelled amphetamine sulphate ip. The pH of the urine remained unchanged by ethanol. With the metabolites isolated by autoradiography on a 2-dimensional chromatograph of urine, amphetamine and its degradation products amounted to 85% of the urine radioactivity, with about 77% of the total radioactivity being recoverable in the first 24 hr. A large increase in the percentage of the radioactivity in the urine as amphetamine, and a decrease in p-hydroxyamphetamine, both free and conjugated, occurred with ethanol pretreatment, the proportion of p-hydroxyamphetamine being excreted in free form and the same in each group. Were amphetamine metabolized by monoamine oxidase, and not by hydroxylation of the benzene ring, then ethanol would be expected to affect its metabolism. Whether this effect of ethanol on amphetamine metabolism is specific or general remains to be answered, as does the question of metabolic interaction between alcohol and amphetamine occurring in man, since the metabolism of amphetamine in man differs from that in the rat.

281. Creaven, P. J., Barbee, T., and Roach, M. K.
THE INTERACTION OF ETHANOL AND AMPHETAMINE METABOLISM.

J. Pharm. Pharmacol. (London), 22(11): 828-831 (17 ref.), 1970.
E – exp. cont. – DC (decrease) – mammals – acute admin. – blood lev. – other drug lev. – absorp., distrib., stor. – metab. proc. – amphetamines – miscellaneous – unclass. ther. agents – *CAAAL-0 B-0920.

The effects of pretreatment with ethanol (1, 3, or 5 g/kg by stomach tube as a 25% sol) on the amount of p-hydroxyamphetamine, 4-hydroxyacetanilide, or 4-hydroxybiphenyl excreted during various time periods after ip injections of 5 mg/kg (\pm)-[2-¹⁴C] amphetamine sulphate, 250 mg/kg acetanilide, or 200 mg/kg biphenyl, respectively, were studied in male Sprague-Dawley rats (100-150). Also determined were the effects on p-hydroxyamphetamine excretion, of ethanol in combination with pyrazole or disulfiram, and of some inhibitors and inducers of liver microsomal oxidation. It was found that ethanol produced a marked depression of hydroxylation of the aromatic ring of amphetamine, the major metabolic pathway of the compound in the rat. The effect was greatest immediately after

ethanol, but was seen 12-24 hr after dosage. Ethanol pretreatment had no effect on acetanilide or biphenyl hydroxylation, suggesting that amphetamine hydroxylation may differ from known microsomal hydroxylations by not being induced by phenobarbitone and carcinogenic hydrocarbons, although it is inhibited by SKF 524-A and other mixed function hydroxylators. Pyrazole greatly enhanced ethanol inhibition of amphetamine hydroxylation, whereas disulfiram did not, indicating that inhibition of amphetamine metabolism is probably mediated through ethanol itself, rather than by the metabolism of ethanol or acetaldehyde.

282. Crémieux, A., Cain, J., and Rabattu, J.
TOXICOMANIE ALCOOLIQUE ET ORTÉDRINIQUE CHEZ UN DÉSÉQUILIBRÉ DE LA SEXUALITÉ. [Alcohol and ortedrine addiction in a sexual deviant].
 Ann. Medicopsychol. (Paris), 106: 497-501 (0 ref.), 1948.
 F – general – conj. addict. – psychot. humans – drug-dep. humans – amphetamines – *CAAAL-5071-L4 A-0628.

A case is described of a 32 yr-old sexually-unbalanced male, dependent upon alcohol since the age of 16. In addition, he had recently begun to take ortedrine in quantities of up to 1.5 g/day. Ortedrine toxicomania, a rare occurrence, coupled with the patient's psychic disturbances, convinced the authors to report their findings.

283. Cruz, I. A., Cramer, N. C., and Parrish, A. E.
HEMODIALYSIS IN CHLORDIAZEPOXIDE TOXICITY.
 J.A.M.A. (Chicago), 202(5): 438-440 (14 ref.), 1967.
 E – SEC – general – case hist. – DC (unspec.) – humans – blood lev. – barbiturates – sed., hypnot. – tranquilizers – *CAAAL-0 B-0269.

Hemodialysis was performed on a woman who had ingested an excessive amount of a number of drugs, including chlordiazepoxide hydrochloride, barbiturates, ethchlorvynol, and alcohol. Before dialysis, the blood levels of these drugs were 2.4 mg/kg, 4.5 mg/kg, 0 mg/kg, and 140 mg/kg, respectively. During dialysis, the plasma level of chlordiazepoxide decreased from 2.4 mg/100 ml to 1 mg/100 ml, which suggests that the drug can be removed in this way from the body. Her ultimate death was felt to be due to overwhelming pulmonary infection.

284. Cummins, J. F., and Friend, D. G.
USE OF CHLORPROMAZINE IN CHRONIC ALCOHOLICS.
 Amer. J. Med. Sci. (Philadelphia), 227: 561-564 (5 ref.), 1954.
 E – general – DC (antidotal) – DC (sensit.) – drug-dep. humans – in vivo – mot. perform. – nerv. syst. – tranquilizers – unclass. ther. agents – *CAAAL-7070-N47 A-1247.

Acute and chronic alcoholics were treated with disulfiram directly upon admission, in contrast to the 6 day alcohol-free period normally observed to avoid a disulfiram-alcohol reaction, and, since it is well known that nausea and vomiting are commonly produced by the ingestion of disulfiram in the presence of alcohol, it was decided to evaluate the effectiveness of chlorpromazine (CPZ) in suppressing such symptoms. Regardless of the amount of liquor previously consumed, patients received 100 mg CPZ and 500 mg disulfiram po immediately, followed 6 hr later by 50 mg CPZ. The disulfiram dose was maintained daily, and the CPZ dosage was decreased until none was given on the fourth day. No untoward side effects were noted in 60 treatment cases. Nausea, vomiting, and hypotensive episodes did not occur, nor was the usual post-alcoholic psychomotor agitation experienced. It is concluded that disulfiram and CPZ, when administered together in adequate dosage to the inebriated alcoholic, produce a tranquilized state more effectively than any current method of sedation.

285. Czerwenka-Wenkstetten, H., Hofmann, G., and Kryspin-Exner, K.
 EIN FALL VON VALIUM-ENTZUGSDELIR. [A case of valium withdrawal delirium].
 Wien. Med. Wschr. (Vienna), 115(47): 994-995 (0 ref.), 1965.
 G – general – case hist. – conj. addict. – psychot. humans – CNS – tranquilizers – *CAAAL-0
 B-0270.

The case of a civil servant who had developed psychic symptoms due to overwork, fatigue and resulting strain, is described. He was prescribed seduan, vesparax, and meprobamate, but also had recourse to alcohol. A chronic alcohol and drug abuse followed, and the man underwent voluntarily a withdrawal regimen in a clinic. The patient recovered quickly and was discharged. Two years later, he relapsed by taking 60-100 mg of valium daily plus alcohol. Upon withdrawal of both, the patient developed unrest, insomnia, and hallucinations. The psychopathological symptoms are described. The patient recovered after two weeks.

286. Czyzyk, A., and Mohnike, G.
 ÜBER DIE BEEINFLUSSUNG DER ALKOHOLTOLERANZ DURCH
 BLUTZUCKERSENKENDE HARNSTOFFDERIVATE. [The effect of blood sugar-lowering
 urea derivatives on alcohol tolerance].
 Deutsch. Med. Wschr. (Stuttgart), 82(36): 1585-1586 (0 ref.), 1957.
 G – exp. comp. – DC (sensit.) – humans – acute admin. – chronic admin. – in vivo – blood lev. –
 metab. proc. – hormones, hormone antag. – *CAAAL-8332-A1 A-1384.

Blood levels of sugar, acetone, alcohol, and acetaldehyde were determined in 6 rabbits on 3 occasions: after administration of 1.5 g/kg alcohol po; after pretreatment for 1 week with 0.5 g BZ-55 (carbutamide)/kg/day po, following which the above alcohol dose then a single dose of 0.1 g carbutamide/kg iv were administered; and after 2 weeks of carbutamide pretreatment (above dosage), followed by the same administrations as on the second occasion. In 5 of the rabbits, alcohol values were the same in experimental and control sessions; in 1 rabbit with very high sulfonamide values, blood alcohol and acetaldehyde were higher after pretreatment. 11 diabetic patients received 1 g/kg alcohol po, and blood samples were taken hourly for 8 hr. The subjects were then pretreated with 1 + 1 + 1 g carbutamide (6 patients) or tolbutamide (6 patients)/day for 7-9 days, after which the same alcohol dose was again given, together with 1 g of the urea compound. Blood samples were taken as before. The blood alcohol curves were the same for control and experimental sessions. Acetaldehyde levels were higher in all patients receiving carbutamide, and in 2 subjects receiving tolbutamide. Acetone levels were slightly higher during the experimental session. It is concluded that high concentrations of the urea derivatives inhibit alcohol dehydrogenase.

287. Dajani, R. M., Ghandour-Mnaymneh, L., and Saadeh, F.
 EFFECT OF THE CONGENERS IN ARAQ ON THE INCIDENCE OF ALCOHOLIC
 FATTY LIVER IN THE RAT.
 Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 34-49 (18 ref.), 1970.
 E – exp. cont. – exp. comp. – congen. stud. – mammals – chronic admin. – in vivo – absorp., distrib.,
 stor. – liver, kidney – *CAAAL-12887-G2 B-0921.

Experiments were conducted to study the effect of congeners present in 2 alcoholic beverages, anisated araq and mastic araq, on formation of alcoholic fatty liver. 80 male rats (100-120 g) were divided into 4 groups. The first group (A), had access to 10% v/v anisated araq. The second group (M) received similarly diluted mastic araq. The third group (E) was given 10% v/v ethanol, and the fourth, control, group received only water. After 6 weeks, the concentration of ethanol was raised to 20% for the remaining 32 weeks of the experiment. Beginning with the introduction of the 20% alcohol, the livers of 2 rats from each group were removed at 2-week intervals, and total lipid, triglyceride, and cholesterol concentrations were determined. At the end of 28 weeks, the average increase in total lipid concentrations for groups E, A, and M were 68, 77, and 98%, respectively, above control values.

Triglyceride levels at this time for E, A, and M groups were 81, 100, and 139%, respectively, above control levels. Total cholesterol levels for groups E, A, and M at 28 weeks were 104, 197, and 278%, respectively, above control values. The data suggest that the araq congeners accentuate the effect of alcohol on fatty liver infiltration. The importance of the observations in the etiology of alcoholic fatty liver, and their probable underlying mechanisms are discussed.

288. Damrau, F., and Liddy, E.

HANGOVERS AND WHISKY CONGENERS: COMPARISON OF WHISKY WITH VODKA.

J. Nat. Med. Ass. (New York), 52(4): 262-265 (13 ref.),

1960.

E – exp. cont. – exp. comp. – congen. stud. – humans – acute admin. – in vivo – cardiovasc. – G.I. tract – senses – *CAAAL-0 A-0629.

The effect of congeners in producing hangover was compared after whiskey and vodka. The group studied included 33 men and 35 women, all non-drinkers or moderate social drinkers and between 22 and 72 yr of age. Each subject at different times drank 2 oz of whiskey or vodka and then answered a questionnaire. The popular belief that vodka causes less hangover than other liquors because of its extremely low congener content was substantiated. Also, the whiskey congeners may have an unfavourable effect in conditioning the response to alcohol; this conditioning was not observed in vodka.

289. Damrau, F., and Liddy, E.

THE WHISKY CONGENERS: COMPARISON OF WHISKY WITH VODKA AS TO TOXIC EFFECTS.

Curr. Ther. Res. (New York), 2(9): 453-457 (8 ref.),

1960.

E – exp. cont. – exp. comp. – congen. stud. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – cardiovasc. – G.I. tract – metab. proc. – senses – *CAAAL-0 A-0630.

Experiments were carried out on 68 male and female subjects to show by organoleptic and chemical methods the actual effect of different congeners on subjects under controlled conditions. The 4 elements of the tests consisted of vodka congeners (1 oz), whiskey congeners (1 oz), vodka (2 oz), and whiskey (2 oz). It was found that whiskey congeners caused a large number of bad taste responses, gastric irritation, vasomotor responses, and a burning effect on the buccal membranes, while vodka caused little or none of the above responses. The whiskey congeners were also found to increase or extend the alcohol effect.

290. Damrau, F., and Liddy, E.

THE USE OF VODKA IN GERIATRICS.

Industr. Med. Surg. (Chicago), 31: 463-464 (10 ref.),

1962.

E – general – congen. stud. – humans – *CAAAL-10256-V3 A-1385.

Although whiskey is the traditional alcoholic beverage prescribed for medicinal purposes, the authors point out the various disadvantages which result from its high congener content. When present in large amounts, the congeners enhance the possibility of toxic reactions. Several published studies conducted by the authors showed that whiskey congeners slowed the rate of oxidation of alcohol, and produced prolonged after-effects, while the same amount of vodka of the same proof failed to produce virtually any hangover symptoms. Similarly, organoleptic responses to whiskey were more severe than to vodka. A psychological study of 100 moderate social drinkers revealed that 83% preferred vodka to whiskey. Vodka, with its relative lack of congeners, is far more advantageous in therapeutic use. More patients prefer the beverage to whiskey, and there is less gastric irritation and fewer hangovers. Furthermore, studies by the authors have indicated that whiskey congeners condition the physiological response to alcohol, whereas vodka does not. This conditioning is one factor which may lead to habituation. Finally, vodka is a pure and uniform beverage, in contrast to whiskey, with its variable and uncertain congener content.

291. Dandiya, P. C., and Rungta, S. S.
 STUDIES ON CENTRAL NERVOUS SYSTEM DEPRESSANTS (VI): CENTRAL
 NERVOUS SYSTEM ACTIONS OF THREE ESTERS OF BENZILIC ACID.
 Arch. Int. Pharmacodyn. (Gand), 149(3-4): 493-506 (19 ref.), 1964.
 E – SEC – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – mot perform. – CNS
 – respir. – analg., antipyret. – *CAAAL-0 A-1386.

The CNS actions of 3 benzilic acid esters— β -dimethyl amino ethyl benzilate hydrochloride (DABH), β -dimethyl amino ethyl ethoxy benzilate hydrochloride (EDABH), and β -dimethyl amino ethyl benzilate octyl bromide (DABOB)—were studied in mice, rats, and dogs. In 1 experiment, pentobarbital sodium (40 mg/kg), hexobarbital (100 mg/kg), and ethanol (4 g/kg) sleeping times in mice were measured, with and without pretreatment with 3 and/or 10 mg/kg of the 3 esters. Both 3 and 10 mg/kg of DABH significantly prolonged pentobarbital sleeping time, as did the 10 mg/kg doses of DABOB and EDABH. 10 mg/kg of all 3 compounds failed to influence hexobarbital or ethanol hypnosis. It is concluded that, since none of the 3 compounds influenced hexobarbital or ethanol sleeping times, the mechanism of their potentiation of pentobarbital hypnosis cannot be entirely attributed to their CNS-depressant action, but may be, at least partly, due to interference with pentobarbital metabolism.

292. Danechmand, L., Casier, H., Hebbelinck, M., and De Schaepdryver, A.
 COMBINED EFFECTS OF ETHANOL AND PSYCHOTROPIC DRUGS ON MUSCULAR
 TONE IN MICE.
 Quart. J. Stud. Alcohol (New Haven), 28(3): 424-429 (16 ref.), 1967.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in
 vivo – mot. perform. – skel., muscle, skin. – tranquilizers – *CAAAL-12439-D2 B-0271.

The tilted-plane test was used to evaluate the effect on the muscle tone of mice, induced by ethanol (1.6-3.2 g/kg) in combination with chlorpromazine (5 mg), meprobamate (100 mg), chlordiazepoxide (3.75 mg/kg), or hydroxyzine (1-2 mg/kg)—all injected ip in a total vol of 0.1-0.2 ml/20 g body wt. Ethanol alone caused loss of tone after 30 min with the 1.6 g/kg dose, after 15 and 30 min with the 2.4 g/kg dose, and after 15, 30, and 45 min with the 3.2 g/kg dose. The other substances caused no change. The 1.6 g/kg ethanol dose, however, caused loss of tone at 15, 30, and 45 min after pretreatment with chlorpromazine, meprobamate, or chlordiazepoxide; hydroxyzine pretreatment induced loss of tone only after 30 min (with the 1 mg/kg dose level). Moreover, when a given dose of ethanol alone did induce a decrease of tone, this decrease was significantly enhanced after pretreatment with the drugs.

293. Danger, W.
 EXPERIMENTELLE STUDIEN ZUR FRAGE DER BEZIEHUNGEN ZWISCHEN
 BLUTALKOHOLGEHALT UND ALKOHOLWIRKUNG. [Experimental studies on the question
 of the relations between the blood alcohol level and alcohol effect].
 Dissertation, Medical Faculty of the University of Göttingen, Germany, 50 pp. (33 ref.), 1938.
 G – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev.
 – mot. perform. – *CAAAL-0 A-0631.

The ring test and the addition test were applied to 5 human subjects in sober condition and after ingestion of 160-400 cc alcohol. Results of the ring test showed that the number of errors multiplied with increasing blood alcohol concentration. Motor functions seemed to be more affected by alcohol than sensory functions. Cigarette smoking was found to enhance the intoxication symptoms subjectively and objectively.

294. DaVanzo, J. P., Ruckart, R., Kang, L., and Daugherty, M.
BIOCHEMICAL STUDIES WITH DOXAPRAM HCL.
 Fed. Proc. (Bethesda), 23 (2, Pt. 1): 386 (0 ref.), 1964.
 E – abst. – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – species or sex diff. – CNS – metab. proc. – analeptics – stimulants – *CAAAL-0 A-1387.

The mechanism of action of doxapram hydrochloride antagonism of ethanol-induced depression was investigated in dogs and rats. Doxapram ($3 \times 10^{-3}M$) stimulated alcohol dehydrogenase (ADH) activity by approximately 100%. Other stimulants at this concentration did not show this degree of stimulation, and 3 drugs—ethamivan, d-amphetamine, and picrotoxin—inhibited ADH activity. Blood alcohol levels after simultaneous administration of doxapram and alcohol were significantly reduced from control values. Doxapram at a concentration of $10^{-5}M$ slightly stimulated cytochrome oxidase and succinoxidase activity. At a concentration of $10^{-3}M$, doxapram failed to inhibit monoamine oxidase activity. Doxapram infused iv resulted in a prompt and marked increase of total blood and urinary catechols in dogs, but did not increase urinary catechols in rats.

295. Davenport, H. W.
GASTRIC MUCOSAL HEMORRHAGE IN DOGS: EFFECTS OF ACID, ASPIRIN, AND ALCOHOL.
 Gastroenterology (Baltimore), 56(3): 439-449 (10 ref.), 1969.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – absorp., distrib., stor. – blood comp., sites, lymph – G.I. tract – skel., muscle, skin – analg., antipyret. – gastrointest. agents – *CAAAL-0 B-0272.

The interrelations between aspirin, hydrochloric acid (HCl), and alcohol were studied with respect to damage of the oxyntic glandular part of the gastric mucosa. The results showed that the mucosa never bled during irrigation with aspirin in neutral sol with or without ethanol. Irrigation with 20 mM aspirin in 1 or 10 mN HCl, with or without ethanol, always caused bleeding. Severe damage and brisk bleeding were always produced by the combination of 20 mM aspirin, 100 mN HCl, and 8-10% ethanol. Sol containing 4-5% ethanol; 10 or 20 mM aspirin; and 1, 10, or 100 mN HCl were more damaging than the corresponding sol containing no ethanol.

296. Davies, E. B.
TRANLYCYPROMINE AND CHEESE.
 Lancet (London), 2(7309): 691-692 (1 ref.), 1963.
 E – SEC – general – DC (unspec.) – humans – cardiovasc. – enzymes – *CAAAL-0 A-0632.

In this letter, attention is drawn to the symptoms of severe headache, nausea, dizziness, and high blood pressure in patients treated with monoamine oxidase inhibitors, who also ingested cheese and/or alcohol. The symptoms are described, and an explanation as to what might cause them is attempted. The author now advises all patients who are taking any monoamine oxidase inhibitors to avoid cheese and alcohol in the diet, particularly large quantities of these foods with an evening meal.

297. Davis, D. A., Yen-Koo, H. C., and Krop, S.
EFFECTS OF CHLORDIAZEPOXIDE (CDP) AND ETHYL ALCOHOL (ETOH) ON LEARNING OF CONDITIONED AVOIDANCE RESPONSE (CAR) IN RATS.
 Fed. Proc. (Bethesda), 30(2): 568Abs (2 ref.), 1971.
 E – abst. – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – psychol. perform. – species or sex diff. – CNS – tranquilizers – *CAAAL-0 B-0923.

The effects of ethanol and chlordiazepoxide (CDP), singly and in combination, on learning of conditioned avoidance response (CAR) were investigated in rats. 25 male and 25 female rats were

divided into 5 groups, each containing 5 rats of each sex. The groups received; no drug (control), saline, ethanol (350-375 mg/kg ip), CDP (5 mg/kg), or ethanol plus CDP (same dosages). The learning ability of the ethanol group was approximately the same as that of controls (15%), while CDP increased CAR in male and female rats to 53% and 38%, respectively. Ethanol plus CDP greatly enhanced learning ability in female rats (to 78%), but only slightly increased CAR beyond the values for CDP alone in male rats. In another trained, poorly-performing group, ethanol and CDP, alone or in combination, caused a 2-fold increase of CAR. It is suggested that ethanol potentiates CDP in relieving anxiety and tension, yet the animals are still alert enough to show high learning ability and retention.

298. Davis, J. H., and Fisk, A. J.

THE DADE COUNTY, FLORIDA, STUDY ON CARBON MONOXIDE, ALCOHOL AND DRUGS IN FATAL SINGLE VEHICLE AUTOMOBILE ACCIDENTS.

National Association of Coroners Seminar, Miami, Florida, 11 pp. (5 ref.), 1966.
E – presentation – stat. surv. – DC (unspec.) – mot. vehic. – post-mort. – blood lev. – CNS – respir. – indust. intox. – *CAAAL-0 B-0273.

From test statistics compiled from fatal single vehicle accidents in Dade County, Florida, data are given concerning 250 alcohol users, 172 drivers tested for carbon monoxide, and 179 tested for drugs. Two cases are given involving the combination of alcohol and carbon monoxide. Only 4% (8) of those drivers tested showed evidence of drug use, and, of these, half had also been drinking. The author notes, however, that statistics were based upon a routine procedure, usually without the aid of other investigative agencies, and a more careful scene and background investigation, followed by additional chemical tests, should reveal a greater incidence of drug intake.

299. Davis, V. E., Walsh, M. J., and Yamanaka, Y.

AUGMENTATION OF ALKALOID FORMATION FROM DOPAMINE BY ALCOHOL AND ACETALDEHYDE IN VITRO.

J. Pharmacol. Exp. Ther. (Baltimore), 174(3): 401-412 (44 ref.), 1970.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vitro – CNS – liver, kidney – metab. proc. – antidepressants – *CAAAL-0 B-0922.

The effects of ethanol and acetaldehyde on the in vitro metabolism of dopamine (DA)-¹⁴C by rat liver and brainstem homogenates were investigated. A 1 ml vol of NAD (16 mmoles), ethanol (100 mmoles), or acetaldehyde (0.5-4.0 mmoles) was added to the incubation media at the expense of phosphate buffer. In the absence of NAD, THP (the alkaloid formed by condensation of dopamine and its corresponding aldehyde) amounted to 55% of the DA deaminated. In liver, ethanol depressed THP production to 34% of the DA deaminated; it enhanced DOPET (3,4-dihydroxyphenylethanol) production, but had little effect on amounts of DOPAC (3,4-dihydroxyphenylacetic acid) or 3,4-dihydroxyaldehyde formed. Acetaldehyde caused a similar (38%) depression of THP formation, in spite of the fact that this metabolite of alcohol inhibited aldehyde dehydrogenase. Addition of NAD attenuated THP formation, but, in the presence of ethanol and added cofactor, THP production was unchanged, while DOPET levels were elevated. Acetaldehyde, in the presence of NAD, significantly augmented THP synthesis. In brainstem homogenates, both ethanol and acetaldehyde potentiated the formation of THP, and decreased conversion of DA to DOPAC. The data suggest that ethanol-induced and aldehyde-mediated modification of dopamine metabolism may contribute to the pharmacologic effects of alcohol.

300. Dawbarn, M., and Le Breton, E.

INFLUENCE DE L'URÉTHANE SUR LA VITESSE D'OXYDATION DE L'ALCOOL CHEZ L'HOMÉOTHERME. [Influence of urethane on the rate of alcohol oxidation in warm-blooded animals].

C.R. Soc. Biol. (Paris), 128: 79-81 (1 ref.), 1938.
 F – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – metab. proc.
 – neoplast. agents – *CAAAL-932-A2 A-0633.

Rabbits and rats received 1.5 g/kg ethanol. After 1 g/kg urethane, the oxidation coefficient changed in rabbits from an average of 271 to 176. In rats, after 0.6 to 1 g/kg urethane, it changed from 322 to 236. It is concluded that, if the temperature of rabbits and rats is kept constant, the ethanol oxidation coefficient is decreased under urethane anesthesia. As the ethanol oxidation coefficient is not changed in rats and mice by barbiturates like somnifene, urethane has a specific inhibiting action.

301. Dawson, W. S.
THE USE OF PARALDEHYDE IN ALCOHOLIC DELIRIUM TREMENS.
 Med. J. Aust. (Sydney), 40(2): 277 (5 ref.), 1953.
 E – general – DC (unspec.) – humans – liver, kidney – analg., antipyret. – autonomic agents – sed.,
 hypnot. – *CAAAL-6848-N6 A-0634.

The author answers statements made in a paper by Steyn, Douw G. ("The use of paraldehyde in alcoholic delirium tremens," Med. J. Aust. (Sydney), 40: 91-93, 1953). He disagrees with Steyn's statements about the dangers of paraldehyde in treatment of acute alcoholism. He considers 2 or 3 drachms of it po or 10 cc im safe and effective, but morphine or hyoscine are dangerous.

302. De Blasi, S., and Storelli, F.
ALCOOL ETILICO E PROCAINA PER VIA ENDOVENOSA COME COADIUVANTI DI ANESTESIA GENERALE. [Intravenous ethyl alcohol and procaine as coadjuvants in general anesthesia].
 Minerva Anest. (Turin), 18: 212-225 (17 ref.), 1952.
 I – general – DC (unspec.) – humans – acute admin. – in vivo – CNS – anesthetics – *CAAAL-0
 A-1388.

In 30 surgical operations of various types, following pre-anesthesia with morphine, atropine, or pantopon plus atropine, 7% ethanol and 0.6-0.8% procaine hydrochloride were infused iv as a physiologic 5% glucose or subtosan sol. Normally about 200 cc of sol were infused in 1/2 hr. Narcosis was then induced with cyclopropane and oxygen, or with pentothal or kemithal, the latter 2 agents being followed by inhalation of cyclopropane and oxygen or nitrous oxide (80%) and oxygen. Usually the alcohol-procaine was stopped after infusion of 500 cc of sol. The total quantity of ethanol given in each case varied from 26-47 cc, total procaine from 1.5-4.5 g, and total vol of sol from 350-650 cc. The results were uniformly satisfactory. A strong analgesic effect was evident in all cases; it was manifested prior to narcosis, and was very pronounced upon awakening and for several hr after the operation. Only small quantities of anesthetics, which, without the alcohol-procaine, would not have had the same effect, were needed. The muscle relaxation induced sometimes necessitated moderate amounts of curariform agents. Intra- and post-operative circulatory conditions and respiration were satisfactory. No untoward complications were noted.

303. Debray, C., Vaille, C., Martin, E., Souchard, M., and Rozé, C.
INFLUENCE DE L'ÉTHANOL SUR LA LITHIASE RÉNALE EXPÉRIMENTALE À L'ÉTHYLÈNE-GLYCOL CHEZ LE RAT. [Influence of ethanol on experimental renal lithiasis induced by ethylene glycol in the rat].
 Presse Med. (Paris), 73: 1559-1561 (33 ref.), 1965.
 F – exp. cont. – exp. comp. – DC (decrease) – chronic admin. – in vivo – dose resp. – blood lev. –
 liver, kidney – metab. proc. – indust. intox. – *CAAAL-11447-D2 B-0274.

Rats in groups of six received, as the only drinking fluid: a) a 1% ethylene glycol (EG) sol; b) a 2%, 4% or 6% ethanol sol; or c) 1% EG with 2%, 4% or 8% ethanol. After 40 days, the rats were

sacrificed. Oxal renal lithiasis developed in 4 rats drinking EG alone, in 4 drinking EG with 2% ethanol, in 2 drinking EG with 4% ethanol, and in no rats given EG plus 8% ethanol. This antagonism between EG and ethanol may be due to an enzymatic competition, in which selective preference for ethanol allows EG to be eliminated, at least in part, in its natural, less toxic form.

304. Degerli, I. U., and Webb, W. R.
 ALCOHOL, CARDIAC FUNCTION, AND CORONARY FLOW.
 Surg. Forum (Chicago), 14: 252-254 (4 ref.), 1963.
 E – SEC – exp. cont. – DC (unspec.) – mammals – acute admin. – in vivo – cardiovasc. – barbiturates
 – *CAAAL-10811-D2 A-1389.

Hemodynamic changes, myocardial function, and coronary flow alterations caused by moderate amounts of alcohol were investigated in 20 healthy mongrel dogs. Anesthesia was induced with minimal dosages of pentobarbital and fractional doses of succinylcholine. Coronary flow, cardiac output, and pressures in the left atrium, right atrium, and femoral artery were monitored. Control values were taken continuously for 15 min, and myocardial function curves obtained. Alcohol (0.5 g/kg in isotonic saline) was administered iv, to give a blood alcohol concentration of 0.07-0.1 mg/100 ml. The decreased myocardial efficiency in dogs under pentobarbital noted by other authors was observed. Coronary flow decreased significantly, and coronary resistance rose, while pulse and peripheral resistance remained unchanged. Cardiac output increased significantly from 2184 to 2804 ml/min within 15 min, then remained the same for the next hr. Function curves 1 hr after alcohol administration always showed marked deterioration, sometimes to the point of cardiac failure. It is concluded that alcohol significantly increases the work load of the heart, while reducing coronary flow and the functional capacity of the myocardium. Susceptibility to shock in an intoxicated state is due to myocardial insufficiency.

305. Delaunois, A. L., and Casier, H.
 INFLUENCE DE L'HYPERTHERMIE SUR LE SORT DE L'ALCOOL DANS
 L'ORGANISME. [Influence of hyperthermia on the fate of alcohol in the organism].
 Arch. Int. Pharmacodyn. (Gand), 68(3-4): 297-310 (36 ref.), 1942.
 F – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – blood lev. – absorp.,
 distrib., stor. – metab. proc. – indust. intox. – *CAAAL-4056-A2 A-0635.

Dogs received alcohol po under different conditions. Some received dinitrocresol sc, before or simultaneously with ethanol, in doses of 3-7 mg/kg body wt. Absorption of ethanol was accelerated by internal and external heat. Maximal concentration occurred earlier in both heat experiments than in controls. Dinitrocresol caused a rise in maximal concentration, and had an influence on elimination of ethanol as well; a dose of 7 mg/kg accelerated elimination, while 3 mg/kg did not influence it.

306. Delay, J., Deniker, P., and Leyrie, J.
 DOSAGE D'ALCOOLÉMIE PRATIQUE CHEZ DES MALADES TRAITÉS PAR LES
 NEUROLEPTIQUES. [Blood alcohol determination in patients treated with neuroleptics].
 Ann. Medicopsychol. (Paris), 120/1: 752-755 (0 ref.), 1962.
 F – exp. – DC (unchanged) – humans – acute admin. – chronic admin. – in vivo – blood lev. –
 antidepressants – sed., hypnot. – tranquilizers – *CAAAL-10238-U1 A-0636.

The "official method" of alcohol determination was used on the blood of 16 patients who had been for a long time (from 45 days to several years) under treatment with neuroleptic drugs at high dosage. The results were 0 in 9 cases, and less than 0.10 g/l in 7. The conclusions of Certhoux J., and Ramet, M. (Ann. Medicopsychol. (Paris), 120/1: 359-364, 1962), that drugs, such as tranquilizers, make blood values found according to the official method of determination erroneous, are thus refuted.

307. Dengler, K., and Glaesel, H. U.
ANWENDUNG VON VALIUM IN DER KLINISCHEN PSYCHIATRIE. [The use of valium in clinical psychiatry].
Med. Welt (Stuttgart), 18(27): 1620-1624 (33 ref.), 1967.
G – SEC – general – DC (antidotal) – drug-dep. humans – CNS – skel., muscle, skin – tranquilizers – *CAAAL-0 B-0275.

204 psychiatric patients suffering from various forms of alcoholism, toxicomania, schizophrenia, depressions, psychopathy, retardation, organic brain damage, and cerebrosclerosis were treated with diazepam. Except for delirium tremens and alcohol hallucinosis, the various forms of alcoholism responded well to the drug. Treatment of alcoholic intoxication consisted of iv injections of diazepam, the dosage on the first day amounting to 20 mg 3 times/day; no incompatibility followed. As an adjuvant to neuroleptics and anti-depressants, diazepam was also successful in the treatment of vegetative symptoms. The drug had also a good muscle-relaxant effect in patients suffering from spasm and rigidity.

308. Dent, J. Y.
ALCOHOLISM AND AMPHETAMINE.
Brit. J. Addict. (London), 44(2): 74 (0 ref.), 1947.
E – general – conj. addict. – DC (decrease) – drug-dep. humans – blood lev. – amphetamines – *CAAAL-4734-M21 A-0637.

The two views that amphetamine a) replaces alcohol, and b) that amphetamine increases craving for alcohol, are both correct, because there are two distinct incentives to drinking: a) persistent low blood pressure—spirits are taken to give this type of person a “lift”, and b) to allay anxiety. To the second type, amphetamine gives no help; it increases their awareness, and they require a further anesthetic against the discomforts of life before gaining relief.

309. Dérobert, and Hadengue
L'INTOXICATION ALCOOLIQUE ÉTHYLO-MÉTHYLIQUE: LE RÔLE PROTECTEUR DE L'ALCOOL ÉTHYLIQUE. [Ethyl-methylic alcoholic poisoning: protective role of ethyl alcohol].
Annales de Médecine Légale, de Criminologie, Police Scientifique, Médecine Sociale et Toxicologie (Paris), 29: 243-245 (6 ref.), 1948.
F – general – DC (decrease) – humans – absorp., distrib., stor. – metab. proc. – amphetamines – *CAAAL-0 A-0638.

The authors offer conclusions based on a study of 17 cases of fatal methanol poisoning and a review of the literature. They content that there is a period of latency which occurs when ethanol is ingested in addition to methanol. When ethanol is taken simultaneously with methanol, the time lapse until the appearance of toxic symptoms corresponds to the time necessary for a nearly complete oxidation of ethanol. When ethanol is absorbed after methanol, but before the onset of methanol intoxication symptoms, the latency period is extended until the ethanol is fully oxidized by the organism. Clinical observations by other researchers have shown that ethanol decreased the intensity of methanol intoxication, when administered at the very onset of poisoning symptoms.

310. Dérot, M., Dérobert, L., Girard, M., Dupeyron, T., and Ménager, M. J.
L'INTOXICATION PAR LE CHLORATE DE SODIUM. [Poisoning with sodium chlorate].
Sem. Hop. (Paris), 24(23): 719-730 (2 ref.), 1948.
F – SEC – DC (add., infra-add., unspec. incr.) – humans – blood comp., sites, lymph – metab. proc. – anti-infectants – *CAAAL-5809-D1 A-0639.

Three cases of poisoning with sodium chlorate are described, and the results of the autopsies of 6 patients are presented. The lethal dose varies individually, due to the varying amounts of methaemoglobin occurring normally in the blood and working as a catalyst in haemoglobin destruction. Alcohol, which produces methaemoglobin, accelerates the intoxication process, and can contribute to the lethal result.

311. Derr, R. F., Aaker, H., Alexander, C. S., and Nagasawa, H. T.
SYNERGISM BETWEEN COBALT AND ETHANOL ON RAT GROWTH RATE.
 J. Nutr. (Philadelphia), 100(5): 521-524 (9 ref.), 1970.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – mammals – chronic admin. – in vivo – other drug lev. – species or sex diff. – blood comp., sites, lymph – cardiovasc. – metab. proc. – unclass. ther. agents – *CAAAL-0 B-0511.

The purpose of this study was to demonstrate that cobalt and ethanol synergistically depress the growth rate of male albino rats and cause significantly smaller hearts containing more zinc. In rat experiments, water was replaced by 10% ethanol, cobaltous chloride sol (1 mg Co/10 ml H₂O) or ethanol plus cobalt. Since the consumption/100 g body weight was less in the combination group, decreased growth rate was not due to increased consumption. Since ethanol increases the tissue NADH/NAD ratio, whereas cobalt irreversibly complexes with the -SH group of dihydrolipoic acid and α -ketoacid oxidase, thereby blocking NAD-dependent reoxidation of dihydrolipoic acid to lipoic acid, it is easy to rationalize how these 2 effects could be more than additive. Although the ratio of heart weight to body weight was less than additive, the fact that cobalt and ethanol independently increased the ratio suggests that both have a deleterious effect on the heart. In combination-treated rats, the heart's zinc content was significantly higher and the heart weight significantly lower than the other groups.

312. Despierres, G., Phelip, H., and Cajgfinger, H.
UNE "ÉPIDÉMIE" DE CRISES ÉPILEPTIFORMES EN MILIEU SANATORIAL: ROLE POSSIBLE DE L'ÉTHYLISME ET DE L'ISONIAZIDE. [An epidemic of epileptiform crises in a sanitarium: possible role of ethylism and isoniazid].
 Poumon et le Coeur (Paris), 11(10): 1041-1048 (17 ref.), 1955.
 F – general – DC (add., infra-add., unspec. incr.) – humans – CNS – antidepressants – *CAAAL-0 A-0640.

Ten epileptic attacks occurring within three months in eight patients in a tuberculosis sanitarium were connected with simultaneous ingestion of isoniazid and alcohol. It was concluded that isoniazid should be given with caution to patients with a history of alcoholism, and that the use of alcoholic beverages should be reduced as completely as possible during such treatment.

313. Deutsch, H.
UNTERSUCHUNGEN ÜBER DIE WIRKSAMKEIT VON LOKALANÄSTHESIERENDEN MITTELN BEI DER GEWÖHNUNG AN ALKOHOL UND HEROIN. [Investigation of the effect of local-anesthetic drugs in cases of tolerance to alcohol and heroin].
 Dissertation, Medical Faculty of the University of Hamburg, Germany, 13 pp. (26 ref.), 1936.
 G – exp. cont. – exp. comp. – DC (unchanged) – mammals – chronic admin. – in vivo – nerv. syst. – analg., antipyret. – anesthetics – *CAAAL-0 A-0641.

The influence of chronic alcohol and heroin administration on cocaine narcosis was studied in rabbits and guinea pigs. 6 or 8 cc/kg of a 25% alcohol sol. was given daily for 8 days (controls remained untreated). Treated and untreated animals received 2 drops of a 0.75% cocaine sol on the cornea daily for 8 days. The cornea reflex was used as a criterion. A shortening or termination of the cocaine anesthesia was not observed. Similarly, it was found that chronic application of heroin to guinea pigs

for 10-12 weeks had no influence on the anesthetic effect of cocaine, novocaine, or pantocaine. An explanation of the discrepancies between present results and those of earlier works is attempted.

314. Devenyi, P., and Wilson, M.

ABUSE OF BARBITURATES IN AN ALCOHOLIC POPULATION.

Canad. Med. Ass. J. (Toronto), 104: 219-221 (1 ref.),

1971.

E – Stat. surv. – conj. addict. – drug-dep. humans – barbiturates – *CAAAL-0

B-0924.

The incidence of barbiturate abuse over a 3-yr period by 893 hospitalized alcoholics is reported. Of 129 patients (15% of total) who abused other drugs, 89 (70% of drug abusers, 10% of total) used barbiturates, alone or with other drugs. Most of the barbiturates used were of the short- and intermediate-acting types, tuinal and seconal being the most common. The barbiturate users tended to be younger than the general alcoholic population (mean age of 40.9, compared to 47.8 for total population), and a larger proportion were female (a male: female ratio of 3:1, compared to 5:1 for total population). Of the barbiturate abusers, 60% were regular users who risked addiction; the maximal daily dose range ascertained from 33 cases indicated that more than half occasionally took more than 800 mg/day. The motive of 71% of the users was to get intoxicated or to augment the effects of alcohol, and 20% took barbiturates to help them to sleep. A history of overdose was given by 38%—14% intentional, 18% accidental, and 6% unknown cause. One third were addicted to such an extent that the drug had to be withdrawn slowly to prevent withdrawal symptoms, and 5 patients had given up alcohol entirely in favour of exclusive barbiturate use. A later prospective survey of 100 alcoholic admissions largely confirmed the findings.

315. Devenyi, P., and Wilson, M.

BARBITURATE ABUSE AND ADDICTION AND THEIR RELATIONSHIP TO ALCOHOL AND ALCOHOLISM.

Canad. Med. Ass. J. (Toronto), 104: 215-218 (42 ref.),

1971.

E – general – conj. addict. – cross-tol. – mot. vehic. – drug-dep. humans – absorp., distrib., stor. – CNS – metab. proc. – barbiturates – *CAAAL-0

B-0925.

The types of barbiturates, barbiturate abuse, treatment of barbiturate withdrawal, alcohol-barbiturate interaction hazards and mechanism of action, and barbiturate cross-tolerance in alcoholics are discussed. Alcoholics are particularly prone to intermittent or continuous barbiturate abuse, which may sometimes amount to complete addiction. Inherent hazards of synergism exist—a substantial number of barbiturate poisonings have been revealed to be accidental, and many need not have been fatal, had alcohol not been present. All experimental data support clinically-accepted observations that alcohol and barbiturates do not mix well. The barbiturates should have no place in treatment of alcoholism, and there is no need for their use in cases in which other drugs are equally effective. The contribution of the medical profession to drug abuse cannot be lightly dismissed. When dealing with known or suspected alcoholics, it is imperative that physicians be judicious in prescribing drugs, and that they avoid barbiturates. One must probe carefully for a history of drug use, and, if there is evidence of drug abuse, particularly barbiturate dependence, treatment should be quickly modified.

316. Dille, J. M., and Ahlquist, R. P.

THE SYNERGISM OF ETHYL ALCOHOL AND SODIUM PENTOBARBITAL.

J. Pharmacol. Exp. Ther. (Baltimore), 61(4): 385-392 (7 ref.),

1937.

E – exp. cont. – exp. comp. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – metab. proc. – barbiturates – *CAAAL-929-A2

A-0642.

The effects of pentobarbital (10 to 15 mg/kg) and alcohol (1.0 to 1.5 g per kg), administered simultaneously iv, were studied in rabbits. A potentiative synergism was found to exist between the two agents. The potentiation of alcohol depression by pentobarbital was greater with small doses. The

average coefficient of potentiation determined for the range of alcohol doses used was as follows: for a dose of 10 mg/kg sodium pentobarbital it was 5.8, for 15 mg it was 4.0, and for 20 mg, it was 3.1. The rate of elimination of alcohol was unaffected by the presence of pentobarbital, and that of pentobarbital was unaffected by alcohol.

317. Di Luzio, N. R.

COMPARATIVE STUDY OF THE EFFECT OF ALCOHOLIC BEVERAGES ON THE DEVELOPMENT OF THE ACUTE ETHANOL-INDUCED FATTY LIVER.

Quart. J. Stud. Alcohol (New Haven), 23(4): 557-561 (17 ref.),

1962.

E – exp. cont. – exp. comp. – congen. stud. – mammals – acute admin. – in vivo – species or sex diff. – absorp., distrib., stor. – blood comp., sites, lymph – liver, kidney – metab. proc. – *CAAAL-9353-B2 A-1273.

Experiments designed to evaluate the effects of commercial alcoholic beverages of varied congener content on plasma and liver lipid alterations were performed. Female rats were fasted for 8 hr, and then groups of 10 were given a single dose po of either saline, isocaloric glucose, or alcohol (6.0 g/kg body wt, 50% sol) in the form of laboratory ethanol, blended whiskey, bourbon, cognac, gin, scotch, or vodka. After 16 hr of fasting, it was found that all groups manifested a similar 10% wt loss, an unaltered hematocrit, similar liver phospholipid, free and ester cholesterol levels, and a uniform elevation of plasma-free fatty acids. Plasma triglycerides, liver wt, and fatty liver development were uniformly increased, and liver triglycerides were increased 3-4 fold only in those groups receiving some form of alcohol. It is concluded that congener content of alcoholic beverages is not a factor in the development of the acute fatty liver, a development which appears to be specifically due to the influence of ethanol on hepatic triglyceride metabolism.

318. Di Luzio, N. R.

EFFECT OF ACUTE AND CHRONIC ADMINISTRATION OF ETHANOL AND ALCOHOLIC BEVERAGES ON TISSUE TRIGLYCERIDES.

Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 26-33 (21 ref.),

1970.

E – exp. cont. – exp. comp. – congen. stud. – mammals – acute admin. – chronic admin. – in vivo – blood lev. – cardiovasc. – blood comp., sites, lymph – liver, kidney – *CAAAL-12886-B2 B-0512.

Experiments were conducted to compare ethanol and congener beverage effects on plasma and liver triglyceride concentration, following acute and chronic administration, to determine the influence of their chronic administration on cardiac triglyceride concentration, and to determine if acute administration of alcoholic beverages modified "intoxication lipemia" induced by simultaneous ethanol-triglyceride administration. Fasted female rats in groups of 9-16 received 3 g absolute ethanol/kg po as a 40% sol or as vodka or bourbon, together with 1 ml corn oil /100 g, and controls received isocaloric sucrose and corn oil. Triglycerides increased 65% in the liver, and sixfold in plasma following acute administration of ethanol-corn oil mixtures. Vodka-corn oil or bourbon-corn oil mixtures induced comparable liver changes, but decreased plasma triglycerides by 52 and 40%, respectively, perhaps by maintaining plasma triglyceride removal mechanisms. In a second (chronic administration) test, average liver triglyceride levels (mg/g) after 3 weeks on a fat-rich diet were: 5.4 with sucrose supplement; 18.0 with ethanol supplement; 30.4 with vodka supplement; and 31.5 with bourbon supplement. No significant change occurred in plasma or heart triglyceride for any group. A 23% mortality rate occurred in the bourbon-supplemented group, but no deaths occurred in the other groups. It is concluded that, "the enhancing effect of alcoholic beverage administration on liver triglyceride administration and on mortality above that induced by ethanol per se may have significance in the pathogenesis and sequels of liver disease in human alcoholics."

319. Dimberg, R.

ALKOHOL OCH PSYKOFARMAKA. [Alcohol and psychopharmacological drugs].

Alkoholfrågen (Stockholm), 56(9): 383-386 (11 ref.),

1962.

S – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – acute admin. – chronic admin. – in vivo – cardiovasc. – tranquilizers – unclass. ther. agents – *CAAAL-0 A-0643.

A series of investigations was carried out in humans to determine the various effects of librium (3 times 10 mg), antabuse (0.8 g), and ethanol (5 cl of 96% alcohol in lemonade), alone and in combination with one another, under both acute and chronic conditions. Some of the results were the induction of a labile blood pressure and hypotonicity with the ingestion of librium plus ethanol (5 hr after ethanol ingestion), and a reaction to the combination of all three drugs similar to that of the ethanol-antabuse combination.

320. Ditman, K. S., and Mooney, H. B.

EFFECTS OF PHENYLTOLOXAMINE IN ALCOHOLICS

Quart. J. Stud. Alcohol (New Haven), 20: 276-280 (3 ref.),

1959.

E – exp. cont. – DC (unspec.) – drug-dep. humans – CNS – autocoids – *CAAAL-8711-M3

A-0644.

26 skid-row alcoholics were treated with 100 or 200 mg tablets of phenyltoloxamine (PRN) or placebo 4 times/day for 4 to 21 days. 8 of the 9 alcoholics who received PRN and could be followed-up complained of fatigue, nervousness and irritability. No such complaints were made by the 12 placebo controls. The incidence of reported drinking was also recorded. In this study, the period of treatment was too brief to determine adequately any possible antidipsotropic effect of PRN, but there was no indication that the drug was at all superior to the placebo in this regard. One patient who was given 100 mg tablets of PRN and continued to drink was given a clinical rating of +2 (fair improvement); on the other hand, a second patient who drank while on the 200 mg PRN medication had a rating of -2 (moderately worse, with some side effects).

321. Ditman, K. S., Moss, T., Forgy, E., Zunin, L., Funk, W., and Lynch, R.

CHARACTERISTICS OF ALCOHOLICS VOLUNTEERING FOR LYSERGIDE TREATMENT.

Quart. J. Stud. Alcohol (New Haven), 31: 414-422 (11 ref.),

1970.

E – SEC – stat. surv. – conj. addict. – med-leg. – drug-dep. humans – psychol. perform. – hallucinogens – sed., hypnot. – *CAAAL-0 B-0513.

168 male alcoholics agreed to participate in a drug study at Viejas Treatment Centre, San Diego California. 99 volunteered for lysergide therapy and appeared, 27 volunteered but failed to appear, and 42 non-volunteers served as controls. The "no-show" group resembled the volunteer group in most characteristics, although, with respect to drug taking, they more closely resembled the non-volunteers, since both these groups denied taking narcotics (as compared to 6% of the volunteer groups), and fewer admitted taking sedatives or marihuana. 2.4% of the non-volunteers and 3.7% of the "no-shows", as compared to 16.2% of the volunteers, admitted taking marihuana; 8 of the 16 marihuana cases in the volunteer group often smoked it while drinking. 11.7% of the non-volunteers, 7.4% of the "no-shows", and 25.3% of the volunteers admitted taking sedatives. The volunteer and "no-show" groups tended to be slightly younger, and had been drinking for slightly fewer years, but had more arrests, both for drunkenness and felonies, than the non-volunteers. Perhaps because the volunteers had been repeatedly in more trouble, and suffered greater distress from their uncontrollable drinking problem than the non-volunteers (who reported feeling more pleasure from their drinking), the former felt a greater need for therapeutic assistance.

322. Divnogorskaia, N. N.
VLIIANIE ETILOVOGO ALKOGOLIA NA MOZGOVOE KROVOOBRAZHENIE.
 [Effect of ethyl alcohol on cerebral circulation].
 Farmakol. Toksik. (Moscow), 23: 256-257 (5 ref.), 1960.
 R – exp. comp. – DC (unspec.) – other org. – acute admin. – in vitro – cardiovasc. – antispasmodics
 – autonomic agents – stimulants – *CAAAL-9201-D2 A-0645.
- Frogs were trepanned, and microscopic observations conducted on the veins of the frontal brain. Following administration of alcohol (4 ml of a 20% sol/100 g), the veins of the soft cerebral membrane became constricted 10 to 15 min later, on the average, by 1/4 of their original diameter. The effect of adrenaline (0.0001 g/100 g), ephedrine (0.05 g/100 g), caffeine (0.01 g/100 g), and papaverine (0.01 g/100 g), on the cerebral veins was vasodilation by about 1/4 to 2/5 of the original diameter. Papaverine administered after alcohol dilated the vessels 20 to 30 min later; ephedrine, adrenaline and caffeine, following the alcohol, caused further constriction of the vessels.
323. Dixon, R. L., and Rall, D. P.
ENHANCEMENT OF ETHANOL TOXICITY BY ETHACRYNIC ACID.
 Proc. Soc. Exp. Biol. Med. (New York), 118: 970-973 (5 ref.), 1965.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – metab. proc. – elect., water-bal. agents – stimulants – *CAAAL-0 B-0276.
- Treatment with ethacrynic acid (100 mg/kg), sc one hr before ip administration of ethanol, was found to increase the lethality of ethanol in mice. Dogs treated with ethacrynic acid (5 mg/kg), iv one hr before 50% ethanol iv (0.789 g/kg), were found to have higher venous blood concentrations than those given alcohol alone. Ethacrynic acid was shown to be a weak in vitro inhibitor of alcohol dehydrogenase. Mechanism of action and possible clinical significance are also discussed.
324. Dobbing, J.
FAECAL BLOOD-LOSS AFTER SODIUM ACETYLSALICYLATE TAKEN WITH ALCOHOL.
 Lancet (London), 1(7593): 527-528 (0 ref.), 1969.
 E – exp. – DC (unspec.) – humans – acute admin. – in vivo – cardiovasc. – G.I. tract – analg., antipyret. – *CAAAL-13401 B-0514.
- The author reinterprets the data presented in the paper by Bouchier and Williams (Lancet, 1(7587): 178-180, 1969) on faecal blood-loss after the combined ingestion of sodium acetylsalicylate and alcohol. Instead of calculating means and standard errors separately for each group of the collection periods, the author suggests that it would be more appropriate to consider the difference between the entire alcohol plus drug period and the entire alcohol plus placebo period for each individual. The results from 4 of the subjects are discarded because of irrelevant bleeding episodes. For the 18 subjects remaining, 18 individual differences are calculated, of which 14 are positive, 3 are negative and 1 is zero. Using this method of statistical analysis, the mean difference is found to be significantly greater than zero. The total extra blood loss caused by taking the drug after alcohol was, on the average, about 0.7 ml with a range of 0-3 ml. It is concluded that aspirin (sodium acetylsalicylate) taken with alcohol produces gastric hemorrhage with significant loss of blood.
325. Dobronravov, P. A.
K VOPROSU O LECHENII ALKOGOLIZMA STRIKHNINOM. [The question of treating alcoholism with strychnine].
 Meditsinskoe Obozrenie (Moscow), 27(9): 886-892 (2 ref.), 1887.
 R – SEC – general – DC (antidotal) – drug-dep. humans – CNS – sed., hypnot. – *CAAAL-0 A-0646.

The effect of strychnine as a therapeutic agent in treating various symptoms of acute and chronic alcoholism is reviewed and discussed. No deaths could be attributed to strychnine therapy. Among the various agents employed, strychnine in doses of 1/30-4/25 g proved most effective in treating intoxication and all other symptoms. In some cases, strychnine was injected in association with chloral (12 hr apart) for epileptic seizures and hallucinations.

326. Doenicke, A.

BEEINTRÄCHTIGUNG DER VERKEHRSSICHERHEIT DURCH BARBITURAT-MEDIKATION UND DURCH DIE KOMBINATION BARBITURAT/ALKOHOL. [Influence of barbiturates and barbiturates combined with alcohol on driving safety].

Arzneimittelforschung (Aulendorf), 12(11): 1050-1054 (15 ref.), 1962.

G – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – barbiturates – *CAAAL-0 A-0002.

52 healthy volunteers received 200 mg butabarbital, and, 6 hr later, 1/2 l beer with an absolute ethanol content of 17-19 g. Several tests were then performed. The tests showed that, up to 24 hr after the barbiturate narcosis, abilities necessary for safe driving were impaired. Alcohol enhanced the effect. Abstinence from alcohol is considered imperative until at least 48 hr after ambulatory iv thiobarbiturate narcosis, and up to 24 hr after application of barbiturates as medication (e.g., sleeping tablets).

327. Doenicke, A., and Sigmund, W.

PRÜFUNG DER VERKEHRSSICHERHEIT NACH VERABREICHUNG VON FLUPHENAZINDIHYDROCHLORID UND ALKOHOL. [Tests for traffic safety after administration of fluphenazine dihydrochloride and alcohol].

Arzneimittelforschung (Aulendorf), 14(8): 907-912 (0 ref.), 1964.

G – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – acute admin. – in vivo – mot. perform. – psychol. perform. – tranquilizers – *CAAAL-0 A-0043.

25 volunteers underwent 7 psychometric tests after placebo, after 1/2 l of beer (17-19 g absolute ethanol content), and after beer and fluphenazine dihydrochloride (1 mg). Results showed that consumption of alcohol, even 8 hr after ingestion of the drug, can lead to impaired concentration, performance, reaction speed, and effective reactions. The authors consider that drug manufacturers should draw attention, not only to the potential impairment of traffic competency from the ingestion of hypnotics and alcohol, but also to the combination of psychotropic agents (day-time sedatives) and alcohol.

328. Doenicke, A, and Kugler, J.

UNTERSUCHUNGEN NACH BARBITURATMEDIKATION UND ZUSÄTZLICHEM ALKOHOLGENUSS IM 24-STUNDEN-VERLAUF: BESTIMMUNG DES BARBITURATSPIEGELS IM SERUM, EEG-KONTROLLE, LEBERFUNKTIONSPROBE UND PSYCHODIAGNOSTISCHE TESTS. [Investigations after barbiturate medication and additional ingestion of alcohol in the course of 24 hours: Establishing the barbiturate level in the serum, EEG control, test of liver function and psychodiagnostic tests].

Aktuelle Probleme der Verkehrsmedizin (Stuttgart), 2: 134-148 (17 ref.), 1965.

G – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – mot. perform. – CNS – anesthetics – barbiturates – sed., hypnot. – *CAAAL-0 B-0277.

Narcoses were induced in volunteers by iv doses of 250 mg, 500 mg, or 1000 mg of thiopental, thiobutabarbital, or thiopental plus halothane or nitrous oxide. 24 hr after the narcosis, the subjects drank 1/2 l of beer (17-19 g of absolute alcohol). The subjects suddenly fell asleep several times during the 24 hr after the injection; after the beer, they were drunk and had difficulty in walking.

329. Doenicke, A., Kugler, J., Spann, W., Liebhardt, E., and Kleinert, H.
 HIRNFUNKTION UND PSYCHODIAGNOSTISCHE UNTERSUCHUNGEN NACH
 INTRAVENÖSEN KURZNARKOSEN UND ALKOHOLBELASTUNGEN. [Brain function
 and psychodiagnostic investigations after intravenous short narcoses and alcohol stress].
 Anaesthesist (Berlin), 15(11): 349-355 (22 ref.), 1966.
 G – ES – exp. comp. – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – acute admin. –
 in vivo – psychol. perform. – sed., hypnot. – *CAAAL-0 B-0278.

250 mg or 500 mg of iv barbiturates tended to induce post-anesthetic sleep in human subjects during the first 12 hr, whereas propanidid or propanidid plus halothane did not. Psychodiagnostic tests during the first 8 hr following the barbiturate showed impaired performance, yet there was no impairment as early as 30-60 min following propanidid (500 mg). Alcohol tests 1 or 2 hr after barbiturate anesthesia showed further impairment in psycho-ability. Alcohol, given 6 hr after methohexital narcosis, showed synergistic action causing drowsiness. Only propanidid was eliminated 30 min after application. Consequently, traffic participation could be permitted 2 hr after propanidid narcosis.

330. Doenicke, A., and Kleinert, H.
 ARZNEIMITTEL, ALKOHOL UND VERKEHRSTÜCHTIGKEIT. [Drugs, alcohol and
 driving efficiency].
 Med. Klin. (Munich), 62(21): 835-840 (33 ref.), 1967.
 G – review – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – humans – psychol.
 perform. – CNS – barbiturates – tranquilizers – *CAAAL-0 B-0279.

The European literature on the topic since 1923 is reviewed. The case material, experiments, and forensic experiences discussed in this paper reveal that drugs are capable of impairing concentration and performance, and that a synergism (additive or potentiative) between barbiturates, psychoactive drugs, or tranquilizers and alcohol is demonstrable by means of psycho-diagnostic tests and the electroencephalogram.

331. Doldt, H.
 FUSELÖLE UND IHRE WIRKUNG AUF DEN TRUNKENHEITSGRAD BEIM GENUSS
 ALKOHOLISCHER GETRÄNKE. [Fusel oils and their effect on the degree of intoxication after
 drinking alcoholic beverages].
 Dissertation, Medical Faculty of the University of Heidelberg, West Germany, 62 pp. (58 ref.), 1964.
 G – exp. cont. – congen. stud. – humans – acute admin. – in vivo – mot. perform. – psychol. perform.
 – senses – *CAAAL-0 A-0647.

Controlled experiments were carried out to determine the toxic effects of congeners in alcoholic beverages administered to healthy subjects. The volunteers were tested for reaction time to light and sound, hand coordination, Romberg's, Bourdon's, bar, writing, and drawing tests. Blood alcohol levels were determined with the Widmark and alcohol dehydrogenase methods. The fusel oil content in the alcohol varied between 0.2% and 1%. The subjects drank 180 to 250 cc of the alcohol mixture. The test with a 14% brandy distillation residue gave slowed-down reaction times and decreased concentration at a blood alcohol level of 0.8‰ Subjective phenomena (headache, drowsiness) persisted for 14-18 hr after the start of the test. Similar results were obtained with 1 1/2 l beer, followed by 2.5 g fusel oil in 250 cc alcohol-free carbonic acid liquid. Tests with pure alcohol showed no change in behaviour of subjects. The higher alcohols had no influence on blood alcohol values.

332. Dolger, H.
 ALCOHOLIC BEVERAGES AND ORALLY GIVEN HYPOGLYCEMIC DRUGS.
 J.A.M.A. (Chicago), 173: 1278 (0 ref.), 1960.

E – general – DC (sensit.) – humans – skel., muscle, skin – anti-infectants – *CAAAL-9248-E3
A-1274.

The author expresses his opinion about the consumption of alcoholic beverages by persons taking oral hypoglycemic drugs. The therapeutic recommendation of “abstinence from either alcohol or sulfonylurea compounds” is thought to be unnecessary, since the purple-red flushing of the skin, or the occasional complication of oral diabetic treatment produced by this combination, is more embarrassing to observers than it is distressing to the patient. It is the author’s experience that administration of any orally-given antihistaminic drug at least 1 hr prior to drinking alcoholic beverages will usually prevent these reactions from occurring in sensitive persons using sulfonylurea compounds.

333. Dubitscher, F.
MEDIKAMENTE UND FAHR SICHERHEIT. [Drugs and driving safety].
 Medizinische Sachverständige (Berlin), 56(6): 131-132 (2 ref.), 1960.
 G – SEC – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – analg.,
 antipyret. – *CAAAL-0 A-0047.

The author cites various cases from his experience in medico-legal practice concerning the effects of dextromoramide and other drugs, and their combination with one another and with alcohol, on driving safety. An example is given in which a cliradon tablet taken prior to, and another taken after ingestion of 2 glasses of beer caused appreciable intoxication symptoms. The author was assured by a tubercular patient that it is a known fact in the sanatorium that tuberculostatic agents enhance the effects of alcohol. Thus, if the patients desire to go out on the town without incurring much expense, they consume large quantities of neoteben, and are able to enjoy a state of pleasant intoxication with very little alcohol.

334. Dubois, R.
**DE L'INFLUENCE DES LIQUIDES ALCOOLICOES [SIC] SUR L'ACTION DES
 SUBSTANCES TOXIQUES.** [On the influence of alcoholic liquids on the action of toxic
 substances].
 Dissertation, Medical Faculty of the University of Paris, France, 113 pp. (0 ref.), 1876.
 F – exp. comp. – DC (antidotal) – DC (decrease) – DC (unchanged) – DC (sensit.) – humans –
 mammals – other org. – acute admin. – in vivo – miscellaneous – *CAAAL-0 A-0648.

The author discusses reports of cases and reported experiments concerning the effects of alcohol on the toxicity of: tobacco, snake venom, certain mushrooms, poisonous fish, antimony, and nitric acid. Experiments were conducted on human subjects, dogs, guinea pigs, hens, and frogs; the substances tested were: arsenic, ammonia, potassium nitrate, sulphuric acid, digitalis, atropine, morphine, strychnine, hydrocyanic acid, and cantharides. The author concludes that alcohol is not a universal antidote for toxic substances, and the antagonism between alcohol and the above poisons is not fully confirmed, although simultaneous injection of alcohol may retard poison action. In certain cases, alcohol may totally check effects of a poison, but the action is parallel rather than antagonistic. Therapeutic doses of alcohol so high as to induce inebriation can cause other complications.

335. Düker, H.
**ÜBER DIE KOMPENSATION VON ALKOHOLWIRKUNGEN DURCH PERVITIN BEI
 FORTLAUFENDER PSYCHISCHER TÄTIGKEIT.** [The off-setting of alcohol effects by
 pervitin during continuous psychic activity].
 Psychologische Beiträge (Meisenheim/Glan), 6: 208-217 (6 ref.), 1961.
 G – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – psychol. perform. – miscellaneous
 – *CAAAL-10245-J1 A-0649.

Arithmetical problems were given to two subjects on eight separate occasions. They drank 300 cc of lemonade, solved problems for 40 min, and then took 6 or 9 mg of methamphetamine, rested for 30 min, and continued with the problems; on four of the eight occasions, they received 30-40 cc of 80% alcohol in the lemonade. Methamphetamine improved the performance in all instances; this was particularly evident on alcohol days when impairment was significant, both in terms of numbers of problems solved and percentage of errors committed. The mean percentage of errors of 11.9% and 15% on alcohol days was reduced by the drug to 4% (6 mg) and 2.2% (9 mg) for 1 subject, and from 6% and 5.9% to 1.5% (6 mg) and 1.6% (9 mg) for the other. On control days, the percentage of errors was 3.8% and 4.5% for 1 subject, and 3.7% and 3% for the other. Subjectively, more effort was required to concentrate after alcohol, and less after methamphetamine.

336. Dumont, E.
 A PROPOSE DE L'IVRESSE AU VOLANT; L'IVRESSE BARBITURIQUE ET LA
 SYNERGIE ENTRE L'ALCOOL ET LES BARBITURIQUES. [On intoxication at the wheel;
 barbiturate intoxication and the synergism between alcohol and barbiturates].
 Revue de Droit Pénal et de Criminologie (Brussels), 39: 348-351 (10 ref.), 1959.
 F – general – DC (add., infra-add., unspec. incr.) – med-leg. – mot. vehic. – humans – mot. perform.
 – CNS – barbiturates – *CAAAL-0 A-1275.

Alcohol and barbiturate intoxications are discussed with respect to driving. The legal blood alcohol limits in some European countries are given. Similarities between the symptoms and effects of alcohol and barbiturate overdoses (slow motion, incoherence of movement, excitation) are noted; the need for careful analysis of the intoxication of an apprehended driver is stressed. References to authors who noted synergistic effects between luminal and alcohol, and between veronal and alcohol, are made, and brief discussion is given on the controversy regarding the possibility of these barbiturates affecting the blood alcohol curve. Synergism or potentiation can take place even when alcohol and barbiturates are taken 7 hr apart—a late sleeping-pill, followed by a glass of wine at lunch, can cause intoxication. It is recommended that drug and alcohol inebriation be equally punishable under the law.

337. Dundee, J. W., and Isaac, M.
 ANTAGONISM TO INTRAVENOUSLY ADMINISTERED ETHANOL BY
 CHLORDIAZEPOXIDE (LIBRIUM).
 In: *Alkohol und Verkehrssicherheit*. Konferenzbericht der 5. Internationalen Konferenz über Alkohol
 und Verkehrssicherheit. [Alcohol and traffic safety: reports of the 5th International Conference on
 Alcohol and Traffic Safety]. Freiburg im Breisgau, West Germany, September 22-27, 1969. Freiburg
 im Breisgau: Hans Ferdinand Schulz Verlag, pp. I.37-I.42 (0 ref.), 1969.
 E – exp. cont. – exp. comp. – presentation – DC (decrease) – DC (add., infra-add., unspec. incr.) –
 humans – acute admin. – in vivo – dose resp. – blood lev. – metab. proc. – barbiturates – *CAAAL-0
 B-0280.

182 female subjects undergoing gynecological operations were divided into groups of 20 or 40 and pretreated with chlordiazepoxide (50 mg im, 100 mg im, or 140 mg im and po), diazepam (10 mg im, or 30 mg im and po), or pentobarbitone (200 mg po), and were then given up to 550 ml 8-10% (w/v) alcohol sol (i.e., 10% v/v or 44-55 g absolute alcohol) as rapidly as possible (usually over 4-5 min). A control group of 40 patients received alcohol alone. It was found that 100 and 140 mg doses of chlordiazepoxide induced a state of resistance to the soporific effect of ethanol, whereas pentobarbitone appeared to augment that effect. Antagonism did not occur after 10 mg diazepam, but it may have occurred after the larger doses. Studies of blood levels showed that the chlordiazepoxide antagonism is associated with an increased brain tolerance to, rather than a more rapid breakdown of, ethanol. Higher blood levels are required to produce sleep after chlordiazepoxide.

338. Dundee, J. W., and Isaac, M.
INTERACTION BETWEEN INTRAVENOUS ALCOHOL AND SOME SEDATIVES AND TRANQUILLIZERS.
Brit. J. Pharmacol. (London), 39(1): 199P-200P (6 ref.), 1970.
E – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans
– acute admin. – in vivo – blood lev. – CNS – barbiturates – tranquilizers – *CAAAL-0 B-0926.

The interaction of alcohol and 5 soporific agents was studied in humans under controlled conditions. Atropine (0.6 mg) was given as premedication, followed by im doses of chlordiazepoxide (100-140 mg), diazepam (10-30 mg), pentobarbitone (100-120 mg), promethazine (50 mg), or cyclizine (50 mg). Alcohol was infused as an 8% w/v sol at 80-150 ml/min as required to a max of 700 ml. Venous blood samples were taken 3-4 min after loss of consciousness. More alcohol was necessary to produce sleep after chlordiaepoxide; this increase was associated with increased concentrations of alcohol in venous blood. Less alcohol was needed to produce sleep after pentobarbitone, although the difference was not significant, and the blood alcohol concentration was significantly less. The effects of the other drugs were insignificant. It is concluded that chlordiaepoxide appears to induce true cerebral tolerance to alcohol, whereas clinical doses of pentobarbitone have the opposite effect.

339. Dundee, J. W., and Isaac, M.
INTERACTION OF ALCOHOL WITH SEDATIVES AND TRANQUILLISERS (A STUDY OF BLOOD LEVELS AT LOSS OF CONSCIOUSNESS FOLLOWING RAPID INFUSION).
Med. Sci. Law (London), 10(4): 220-224 (17 ref.), 1970.
E – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans
– acute admin. – in vivo – blood lev. – CNS – barbiturates – tranquilizers – *CAAAL-0 B-0927.

The interaction of alcohol anesthesia with various soporifics was investigated in healthy female humans scheduled for minor gynecological operations. In addition to 0.6 mg atropine, the following drugs were given as premedication 60-90 min prior to anesthesia: pentobarbitone (100 mg im, with or without 100 mg po on morning of operation), chlordiaepoxide (50 mg im), chlordiaepoxide (100 mg im, usually with 20 mg po on night before and again on morning of operation), diazepam (10 mg im, with 10 mg po on night before and again on morning of operation), diazepam (10 mg im, with 10 mg po on night before and on morning of operation), promethazine (50 mg im), or cyclizine (50 mg im). Alcohol in 8% w/v sol was rapidly infused iv in doses sufficient to produce sleep or to a max of 550 ml (44 g), except with the 100-140 mg dose of chlordiaepoxide, when the max alcohol dose was 800 ml. After sleep was induced, anesthesia was continued with 75% nitrous oxide in oxygen. Forearm venous blood alcohol concentrations determined 3-4 min after loss of consciousness revealed that the mean alcohol concentration was significantly less after pentobarbital than in control patients given no soporific premedication. The sleep-onset alcohol level was very significantly increased by the 100-140 mg chlordiaepoxide dose, but was unaffected by the 50 mg dose, or by any of the other premedicants. It is concluded that chlordiaepoxide is an alcohol antagonist, but no explanation is offered for the finding.

340. Dundee, J. W., and Isaac, M.
INTERACTION BETWEEN INTRAVENOUS ALCOHOL AND SOME SEDATIVES AND TRANQUILLISERS.
Med. Sci. Law (London), 11(1): 49-50 (6 ref.), 1971.
E – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans
– acute admin. – in vivo – blood lev. – CNS – barbiturates – tranquilizers – *CAAAL-0 B-0928.

The interaction of alcohol and 5 soporific agents was studied in humans under controlled conditions. Atropine (0.6 mg) was given as premedication, followed by im doses of chlordiaepoxide (100-140 mg), diazepam (10-30 mg), pentobarbitone (100-120 mg), promethazine (50 mg), or cyclizine (50 mg). Alcohol was infused as an 8% w/v sol at 80-150 ml/min as required to a max of 700 ml. Venous

blood samples were taken 3-4 min after loss of consciousness. More alcohol was necessary to produce sleep after chlordiazepoxide; this increase was associated with increased concentrations of alcohol in venous blood. Less alcohol was needed to produce sleep after pentobarbitone, although the difference was not significant, and the blood alcohol concentration was significantly less. The effects of the other drugs were insignificant. It is concluded that chlordiazepoxide appears to induce true cerebral tolerance to alcohol, whereas clinical doses of pentobarbitone have the opposite effect.

341. Duritz, G., and Truitt, E. B., Jr.
IMPORTANCE OF ACETALDEHYDE IN THE ACTION OF ETHANOL ON BRAIN NOREPINEPHRINE AND 5-HYDROXYTRYPTAMINE.
 Biochem. Pharmacol. (New York), 15: 711-721 (50 ref.), 1966.
 E – exp. cont. – DC (unchanged) – DC (sensit.) – mammals – acute admin. – in vivo – CNS – metab. proc. – nerv. syst. – tranquilizers – unclass. ther. agents – *CAAAL-0 B-0281.

The effects of ethanol alone (2 or 4 mg/kg) produced no significant decreases in rat and rabbit brain norepinephrine (NE) and 5-hydroxytryptamine (5-HT) concentrations. However, increased acetaldehyde blood levels produced by disulfiram pretreatment (200 mg/kg) before the ethanol doses, or by the administration of acetaldehyde itself (300 mg/kg), caused statistically significant decreases in brain NE, but no further effects on 5-HT. Chlorpromazine (10 mg/kg) blocked decreases of both 5-HT and NE, without producing any change in the blood levels of acetaldehyde or ethanol.

342. Eckart, D. E.
SODIUM PENTOTHAL SOLUTION, AN ADJUVANT IN THE TREATMENT OF ACUTE ALCOHOLISM.
 J. Kansas Med. Soc. (Topeka), 55: 453-454 (10 ref.), 1954.
 E – SEC – general – case hist. – DC (unspec.) – humans – blood lev. – CNS – glands – metab. proc. – anesthetics – elect., water-bal. agents – hormones, hormone antag. – *CAAAL-7129-N9 A-1248.

The author recommends that, upon admission to hospital, especially if a large previous alcohol intake is suspected, patients in an acute alcoholic state be put to sleep with 10-15 cc 2 1/2% sodium pentothal, and then given a sol of 1000 cc of 10% glucose and water or saline containing 2 g sodium pentothal, iv. 10-15 units of insulin may also be given sc upon admission, or, if the sodium pentothal is stopped 2 1/2-3 hr before breakfast, 15-50 units of insulin may be given 1 hr prior to a breakfast including sweetened fruit juices. The 0.2% sodium pentothal sol is then given only during the night hr (7:30-11:30 pm) until the third day, and then continuously during the third to fifth days, when development of delirium tremens or toxic psychosis is most likely. The author concludes with his opinion of the action of alcohol on the adrenals and the CNS, and the underlying therapeutic mechanism of sodium pentothal-glucose combined with insulin.

343. Editorial
ALCOHOL AND BARBITURATES.
 Brit. Med. J. (London), 1:1269-1270 (11 ref.), 1953.
 E – review – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – blood lev. – absorp., distrib., stor. – CNS – barbiturates – *CAAAL-6498-D3 A-0650.

The literature on the effects of the combined use of alcohol and barbiturates is reviewed. Observations on humans and experimental animals suggest that synergism, if not potentiation, may occur. At present, vast quantities of alcohol are consumed, and, in the U.S.A. in 1948, 300 tons of barbiturates were administered. Fatalities from the combined effect are very rare in relation to the size of the population exposed to the risk. Patients receiving large doses of barbiturates should certainly be warned about taking alcohol, and the possible impairment of driving ability by small doses of each in combination should be stressed.

344. Editorial

ALKOHOL UND MEDIKAMENTE. [Alcohol and drugs].

Blutalkohol (Hamburg), 5: 360-361 (0 ref.),

1968.

G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – acute admin. – in vivo – mot. perform. – CNS – barbiturates – sed., hypnot. – tranquilizers – *CAAAL-0 B-1008.

The results, released by the Swiss Council for Accident Prevention, of a study on the effects of drugs and drug-alcohol combinations on driving ability, are reported. 200 volunteers from the Basel police force were assigned a driving test, while reaction time and the incidence of errors were recorded. Following this, the subjects received 1 of the following administrations po: 20 mg chlordiazepoxide, 800 mg meprobamate, 200 mg phenobarbital, 200 mg methypylone, or a placebo. In addition, half of the subjects received sufficient alcohol in the form of white wine to achieve a blood alcohol concentration of 0.8°/oo within 80 min. The driving test was repeated 3 times—1/2 hr after the alcohol, 2 hr after the drug, and 6 hr after the drug + alcohol (the last test for determining after-effects). The number of errors was somewhat increased by chlordiazepoxide and meprobamate, and slightly more increased by phenobarbital and methypylone. After alcohol, the increase in the number of errors was strongly marked and highly significant, and, with the combined administration, an increase in the alcohol effect was noted with all 4 medications. reaction time was not significantly affected by any administration. The hazards of simultaneous ingestion of alcohol and tranquilizers or hypnotics are stressed. Also reported in Kielholz, P., et al. (Deutsch. Med. Wschr., 94(7): 301-306, 1969).

345. Editorial (Annotations)

ASPIRIN, ALCOHOL, AND GASTRIC HAEMORRHAGE.

Lancet (London), 1(7587): 196 (11 ref.),

1969.

E – general – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – mammals – absorp., distrib., stor. – acid-base, blood pH, elect. – G.I. tract – analg., antipyret. – *CAAAL-0 B-0282.

Some factors concerned with the association of aspirin, alcohol and gastric haemorrhage are discussed. Observations in animals and man have shown the following factors to be important: duration of contact, contact area, and degree of ionization. Reference to previous experiments, in which buffered preparations did not increase faecal blood loss in healthy male volunteers, is made. It was also found that neither the taking of a moderate quantity of alcohol nor the interaction of alcohol and salicylate increased blood loss.

346. Editorial (Annotations)

BARBITURATES AND ALCOHOL.

Lancet (London), 264: 1140 (7 ref.),

1953.

E – general – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – med.-leg. – post-mort. – humans – mammals – CNS – barbiturates – *CAAAL-6898-D1 A-0651.

The question whether alcohol potentiates the barbiturates is discussed. Two recent deaths from barbiturate poisoning after combined intake of barbiturates and alcohol are compared with previously-published animal experiments which demonstrated synergistic action, but no potentiation. The fact that barbiturates are in themselves deadly drugs, and that their effects vary considerably from person to person, is emphasized. The editorial concludes that the dangers of giving barbiturates to those who take alcohol have probably been overstated. It is pointed out that, "thousands of people take barbiturates in the usual doses, even more drink alcohol in one form or another, and yet few deaths have been proved to be due to the two drugs in combination." The 2 deaths referred to may well have been examples of particular idiosyncrasies.

347. Editorial

CONJUNCTIVE USE OF ALCOHOL, DRUGS IS CITED AS INCIPIENT STATE PUBLIC HEALTH PROBLEM: WILBAR APPEAL FOR RESEARCH AND EDUCATION REFLECTS URGENT NEED TO EXPLORE, EXPOUND DEADLY 'POTENTIATION' OF DOUBLE ADDICTION.

Target (Harrisburg, Pa.), 16(1): 1-4 (0 ref.),

1966.

E – general – review – conj. addict. – DC (add., infra-add., unspec. incr.) – drug-dep. humans – barbiturates – sed., hypnot. – tranquilizers – *CAAAL-0 B-0283.

This editorial emphasizes the dangers of combining drugs with alcohol. Although barbiturates were found to be the most commonly abused drugs in combination with alcohol, the tranquilizers came a close second in a 1961 survey made in Philadelphia. In a study made in 1964 (Kaye, Sidney, and Haag, Harvey B., *Toxic. Appl. Pharmacol.*, 6: 316-320, 1964), paraldehyde, considered a safe drug for over 80 yr in treating acute alcoholism, was implicated in the deaths of 9 patients treated for alcoholism. The irony of the alcohol-drug potentiation is thus manifested. "The drugs that are used in the alcoholic's acute phase become the very instruments of a more serious complication of his disease. The alcoholic patient has in some cases become dependent on the very drug used to alleviate his suffering." It is concluded that the injudicious use of alcohol and any drug represents a real threat to those suffering from any form of emotional disorder.

348. Editorial

DRUGS AND DRIVING.

Traffic Laws Commentary (Washington), No. 65-1: 1-17 (97 ref.),

1965.

E – SEC – review – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev. – CNS – barbiturates – *CAAAL-0 B-0284.

This review contains a comparison of U.S. state laws with code provisions on drugs and driving, including recent amendments on driving under the combined influence of alcohol and a drug. It is prefaced with a general review of various drugs (narcotic and non-narcotic) affecting the central nervous system, their conventional uses, the possible impairing effects they can have on physical and mental functioning, and the dangers of possible synergistic effects between various drug combinations.

349. Editorial

EFFEKTEN AV ALKOHOL + MEDICIN: OVÄNTADE REAKTIONER MÖJLIGA.

[Effects of alcohol + drugs: unexpected reactions are possible].

Alkoholfrågan (Stockholm), 61(4): 181-183 (0 ref.),

1967.

S – general – DC (add., infra-add., unspec. incr.) – humans – absorp., distrib., stor. – CNS – metab. proc. – anti-infectants – enzymes – miscellaneous – sed., hypnot. – stimulants – unclass. ther. agents – *CAAAL-0 B-0285.

This is a reprint of an article by Matti K. Paasonen in *Alkoholpolitik*, 29(3): 103-106, 1966. Possible alcohol-drug combinations are reviewed. Discussed are: drugs depressing the central nervous system and whether they cause synergism or potentiation of the effects of alcohol, drugs affecting alcohol metabolism, and the influence of alcohol on drug absorption. Drug combinations should be used with great caution.

350. Editorial

LE MÉPROBAMATE AGGRAVE LES EFFETS DE L'INGESTION D'ALCOOL.

[Meprobamate augments the effects of alcohol ingestion].

Presse Med. (Paris), 68(55): 2144 (2 ref.),

1960.

F – general – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – psychol. perform. – tranquilizers – *CAAAL-0 A-0652.

This editorial refers to recent (*circa* 1960) studies on chlorpromazine- and meprobamate-alcohol interaction—Kopmann, Ernst, and Hughes, Francis W. (Arch. Gen. Psychiat. (Chicago), 1: 7-11, 1959); Marquis, Donald G., et al. (Ann. N.Y. Acad. Sci. (New York), 67: 701-711, 1957); Zirkle, George A., et al. (J.A.M.A. (Chicago), 171: 1496-1499, 1959); and, especially, the study by Zirkle, George A., et al. (J.A.M.A. (Chicago), 173: 1823-1825, 1969) on the effects of meprobamate and small amounts of alcohol on human ability, coordination, and judgement. The 1960 study by Zirkle, et al. is discussed at length. The conclusions of the above experiments show that it is necessary to warn patients treated with meprobamate against the danger of alcoholic drinks, particularly when they have to drive a car or operate complex machines.

351. Editorial

PENNSYLVANIA DEPARTMENT OF HEALTH NOTES DANGERS OF CONJUNCTIVE USE OF ALCOHOL AND DRUGS.

Maryland Med. J. (Baltimore), 16: 134-139 (0 ref.), 1967.
E – general – review – conj. addict. – DC (add., infra-add., unspec. incr.) – drug-dep. humans – barbiturates – hallucinogens – sed. hypnot. – tranquilizers – *CAAAL-0 B-0286.

The editorial appearing in Target (Harrisburg, Pa.), 16(1): 1-4, 1966, is reprinted. This editorial emphasizes the dangers of combining drugs with alcohol. Although barbiturates were found to be the most commonly abused drugs in combination with alcohol, the tranquilizers came a close second in a 1961 survey made in Philadelphia. In a study made in 1964 (Kaye, Sidney, and Haag, Harvey B., Toxic. Appl. Pharmacol., 6: 316-320, 1964), paraldehyde, considered a safe drug for over 80 yr in treating acute alcoholism, was implicated in the deaths of 9 patients treated for alcoholism. The irony of the alcohol-drug potentiation is thus manifested. "The drugs that are used in the alcoholic's acute phase become the very instruments of a more serious complication of his disease. The alcoholic patient has in some cases become dependent on the very drug used to alleviate his suffering." It is concluded that the injudicious use of alcohol and any drug represents a real threat to those suffering from any form of emotional disorder.

352. Editorial (Nouvelles des Sciences)

SPÉCIFIQUE CONTRE L'IVRESSE. [Specific remedy for drunkenness].

Journal de Pharmacie et des Sciences Accessoires (Paris), (Ser. 2) 7: 348 (0 ref.), 1821.
F – review – DC (antidotal) – humans – G.I. tract – CNS – stimulants – *CAAAL-0 A-1276.

This editorial reports on the successful use by Dr. Girard (see: Journal Général de Médecine, 73: 166-178, 1820) of liquid ammonia as a cure for inebriation (which he considers a nervous disorder). 7 to 8 drops diluted in half a glass of water are enough to produce the desired effect. Dr. Chantourelle (see: Journal Général de Médecine, 73: 178-183, 1820) has found by chemical analysis that this is not due to any chemical decomposition of the wine by the ammonia; rather, he believes that the ammonia modifies the sensitivity of the mucous membrane of the stomach and acts on the nerves there, which then transmit to the brain the stimulus they have received.

353. Editorial

TRAITEMENT DE L'EMPOISONNEMENT PAR L'ALCOOL. [Treatment of alcohol poisoning].

Revue Médicale (Paris), 3: 134-135 (0 ref.), 1895.
F – general – DC (antidotal) – humans – cardiovasc. – CNS – respir. – autonomic agents – cardiovasc. agents – elect., water-bal. agents – stimulants – *CAAAL-0 A-0653.

For various types of alcohol intoxication, the recommended treatment is a mixture of trinitrin 1/50 grain (1 mg), atropine 1/100 grain (1/2 mg), and tincture of digitalis sc. A tube is introduced through the mouth or nose (in case of obstacles) into the stomach. Tepid water in liberal quantities is used

for stomach lavage, and a mixture of warm milk and oil (120 g of each), to which 4 g sodium chloride is added in a mixture with 2 laxatives, is offered to the patient. One hr later, 2 mg strychnine sulfate are administered sc. The injection may be renewed three hr after cardiac contraction is lifted.

354. Edmondson, H. A., Hall, E. M., and Myers, R. O.

PATHOLOGY OF ALCOHOLISM.

In: Thompson, G. N., ed. *Alcoholism* Springfield: Charles C. Thomas,

pp. 233-239 (160 ref.) @ 1956.

E – SEC – review – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – CNS – G.I. tract – liver, kidney – metab. proc. – anesthetics – miscellaneous – *CAAAL-0 A-1390.

The author discusses the effects of acute alcoholism on the gastrointestinal tract, liver, pancreas, spleen, hematopoietic system, immunity, adrenals, urinary system, cardiovascular system, and nervous system. Also discussed are the pathology of chronic alcoholism, and its effects on the nervous system and digestive tract. It is noted that even small amounts of alcohol can markedly increase the toxicity of carbon tetrachloride, and can aggravate the toxic action of phosphorus, nitroglycerine, ether, nitrobenzene, aniline, lead, and mercury. Alcohol is contraindicated after phenol ingestion, because the solvent action will increase absorption from the gastrointestinal tract. The synergism of alcohol with carbon disulphide and the barbiturates is mentioned, the practical importance of the latter interaction being stressed, due to its frequent occurrence in medico-legal practice.

355. Eerola, R., Venho, I., Vartiainen, O., and Venho, E. V.

ACUTE ALCOHOLIC POISONING AND MORPHINE: AN EXPERIMENTAL STUDY OF THE SYNERGISM OF MORPHINE AND ETHYL ALCOHOL IN MICE.

Ann. Med. Exp. Biol. Fenn. (Helsinki), 33: 253-261 (6 ref.),

1955.

E – exp. comp. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – dose resp. – G.I. tract – hallucinogens – *CAAAL-7508-D2 A-0654.

The toxic effects of simultaneously-administered alcohol (2% sol injected sc) and morphine (20% sol directed into the stomach by a cannula) were studied in white mice. A potentiative type of synergism was shown between ethanol and morphine. The potentiation was more distinct with small than with large doses. In cases in which the additive mortality would have been 10-25% with small doses, it was nearly three times higher. With the largest doses, the mortality was approximately twice that of additive mortality.

356. Eerola, R.

THE EFFECT OF ETHANOL ON THE TOXICITY OF HEXOBARBITAL, THIOPIENTAL, MORPHINE, ATROPINE AND SCOPOLAMINE: AN EXPERIMENTAL STUDY ON MICE.

Ann. Med. Exp. Biol. Fenn. (Helsinki), 39(Suppl. 3): 1-70 (140 ref.),

1961.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – autonomic agents – barbiturates – sed., hypnot. – *CAAAL-0

A-0655.

The literature on the toxicity and the combined action of the above drugs, and previously used methods, is extensively reviewed. The final results of experiments with some 2900 mice revealed the existence of additive synergism between all the combinations of the drugs examined, when given in sublethal or lethal doses, and ethanol. The combined effect of ethanol and morphine came closest to the algebraic sum. For no combinations of the drugs was the combined graph column half the height of the column which denoted the individual effects of the respective drugs, nor did the area of the combined action fall inside the connecting line in any co-ordinate diagram. The latter would have indicated a potentiative synergism. The experimental results are not directly applicable to man.

357. Eerola, R.
 THE EFFECT OF ETHANOL ON THE TOXICITY OF PROMAZINE, CHLORPROMAZINE, PROMETHAZINE AND HYDROXYZINE: AN EXPERIMENTAL STUDY ON MICE.
 Acta Anaesth. Scand. (Aarhus), 7: 87-95 (15 ref.), 1963.
 E – GS – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – sed., hypnot. – tranquilizers – *CAAAL-10664-D2 A-0656.
- Alcohol (7 mg/kg) was given in 20% concentration, and the drugs in 2.5% concentration, to a total of 1,800 mice. The alcohol was given alone; promazine (400 mg/kg), chlorpromazine (300 mg/kg), promethazine (400 mg/kg), and hydroxyzine (400 mg/kg) were given singly, and then, in the same dosages, combined with alcohol in the same dosage. An additive synergism of all combinations of drugs was shown.
358. Eerola, R., and Alha, A.
 SYNERGISMUS VON ALKOHOL UND BERUHIGUNGSMITTELN. [Synergism of alcohol and sedatives].
 Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 53: 201-210 (33 ref.), 1963.
 G – stat. surv. – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – blood lev. – other drug lev. – alcohols – barbiturates – sed., hypnot. – *CAAAL-10647-D1 A-0657.
- Of toxic cases treated in the Institute of Forensic Medicine, University of Helsinki, in the years 1949-1960, 59 cases which involved a lethal synergism between sedatives and alcohol are presented in tables. Ethanol was ingested in 50 cases, methanol in 2, and both in 7. The sedatives used were mostly barbiturates, although morphine was a contributory factor in 10 cases. All died 30 min-4 hr after taking the sedatives. Blood alcohol levels at the time of death were between 0.9 and 3.07‰. 5 case histories are presented. In all cases, a synergistic effect was shown.
359. Eerola, R., and Alha, A.
 FATAL POISONING WITH CERTAIN DRUG COMBINATIONS.
 J. Forensic Sci. (Mundelein), 9(1): 53-62 (21 ref.), 1964.
 E – stat. surv. – DC (add., infra-add., unspec. incr.) – humans – blood lev. – cardiovasc. – liver, kidney – respir. – sed., hypnot. – tranquilizers – *CAAAL-0 A-0658.
- An investigation of fatal drug poisonings in Finland between 1950 and 1960 showed that the number of annual poisonings had increased by 1960. In 1950, 60 cases occurred—77% one drug, 17% involving two drugs, and 6% three drugs. In 1955, 122 cases occurred—60% involving one drug, 30% two drugs, 7% three drugs, and 3% more than three drugs, and, in 1960, there were 160 cases—46% involving one drug, 30% two drugs, 14% three drugs, and 10% more than three drugs. The 87 cases of 1960 which showed the presence of 2 or more drugs formed the basis of the present study. The cases are analyzed according to case histories, autopsy reports and pathological-anatomical findings, results of chemical analyses for drugs, and the time interval between drug ingestion and death. Synergism between sedatives and alcohol was definitely established in 13 cases.
360. Eggleton, M. G.
 SOME FACTORS AFFECTING THE METABOLIC RATE OF ALCOHOL.
 J. Physiol. (London), 98: 239-254 (18 ref.), 1940.
 E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – liver, kidney – metab. proc. – barbiturates – *CAAAL-2963-A2 A-0659.
- Using mainly cats, the author attempted to determine the effects on the metabolism of alcohol of: (1) the concentration of alcohol, (2) the size of liver, (3) previous feeding with alcohol, and (4) the

amino acid, alanine. Various observations indicated that deep nembutal anaesthesia reduced the metabolic rate of alcohol, and suggested the possibility that even the light degree of anaesthesia normally used in these experiments might have affected the results. The work of several researchers on this problem is investigated.

361. Eggleton, M. G.
THE EFFECT OF NICOTINE ON THE DIURESIS INDUCED BY ETHYL ALCOHOL.
 J. Physiol. (London), 108: 482-490 (8 ref.), 1949.
 E – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – mot. perform. – liver, kidney – nerv. syst. – *CAAAL-5290-D1 A-1391.

Nicotine tartrate (1 mg sc) was given to 22 male and female humans 5-15 min before or after ingestion of alcohol (usually 32 g in 200 cc), and the course of diuresis and total urine output in 2.5 hr were compared in each subject with data from a similar experiment conducted on the same subjects (in most cases, 7 days previously), in which alcohol was given without nicotine. Nicotine caused a decreased urine output of from 6-83% in 11 of 12 smokers, but no such diminution was observed in 10 non-smokers (in 4 of whom an increase occurred). This difference in response of the 2 groups could not be attributed to a differential effect on either the rate of alcohol absorption or the glomerular filtration rate. The explanation is offered that, whereas nicotine stimulation of the sympathetic system antagonized the output of antidiuretic hormone in non-smokers, it caused an increased output of the hormone in smokers, as indicated by an increased lag in onset of diuresis.

362. Ehring, F.
VERKEHRSGEFÄHRDUNG DURCH INH UND ANDERE MEDIKAMENTE VOM STANDPUNKT DES DERMATOLOGEN. [Traffic danger posed by INH and other drugs, from the standpoint of dermatologists].
 Med. Klin. (Berlin), 50(23): 979-980 (40 ref.), 1955.
 G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – absorp., distrib., stor. – metab. proc. – analg., antipyret. – anti-infectants – barbiturates – elect., water-bal. agents – *CAAAL-0 A-1392.

The author emphasizes the possibility of hazardous side effects of certain commonly-prescribed drugs, particularly after alcohol ingestion. Large doses (8-15 mg/kg/day) of isoniazid (INH) taken by patients with tuberculosis and lupus erythematosus can cause exhaustion, euphoria, restlessness, loss of memory, and other disorders. In healthy subjects, alcohol consumption after INH ingestion can cause a pathological intoxication; similar symptoms are observed with irgapyrin, saridon, pyramidon, cibalgil, luminal, and cortisone. When treating patients with lupus erythematosus, such effects are noted with atebirin, resorcin, and cortisone. It is concluded that, although these side effects may present no immediate danger in treatment, prolonged use may cause harm, and the possibility of an untoward reaction with alcohol, especially with regard to the effect on driving ability, is stressed.

363. Eicke, W. -J.
ZWISCHENFÄLLE BEI EINNAHME VON MEPROBAMAT UND GLEICHZEITIGEM GENUSS VON ALKOHOL. [Incidents during treatment with meprobamate and simultaneous consumption of alcohol].
 International Pharmacopsychiatry (Basel), 3: 203-212 (21 ref.), 1969.
 G – ES – general – case hist. – DC (add., infra-add., unspec. incr.) – mot. vehic. – psychot. humans – mot. perform. – psychol. perform. – CNS – G.I. tract – cardiovasc. – senses – tranquilizers – *CAAAL-0 B-0515.

The author states that, although the potentiating action of tranquilizers with alcohol is often discussed, in only 1 instance has an actual case been cited in the literature (see: Torka, Johanna,

Munchen. Med. Wschr., 103: 896, 1961). 3 case histories are briefly described, the subjects all being psychologically labile individuals. Upon taking several tablets of meprobamate and some alcohol, 2 persons experienced extreme intoxication with subsequent amnesia, the third experienced sensory illusions, sweating, nausea, and vomiting. The literature on the subject is reviewed, and it is pointed out that meprobamate changes electroencephalograph readings in a manner similar to barbiturates, and reduces the activity of the hypothalamus. Some authors state that driving ability is not affected by 400 g meprobamate plus up to .8°/oo alcohol, or by 800 g meprobamate alone; others contend that 400 g meprobamate plus .5°/oo alcohol greatly reduce capability. The author concludes that the combined effect of meprobamate and alcohol depends also upon the instability of the personality involved, something which few writers have considered.

364. Eickstedt, K. -W. von

DIE BEEINFLUSSUNG DER ALKOHOL-WIRKUNG DURCH VALEPOTRIATE.

[Modification of the effect of alcohol by valepotriates].

Arzneimittelforschung (Aulendorf), 19: 995-997 (11 ref.),

1969.

G – ES – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – CNS – sed., hypnot. – tranquilizers – *CAAAL-14219

B-1009.

The influence of valepotriates from *Valeriana* root on the hypnotic and narcotic effects and toxicity of alcohol was investigated in mice. Rotarod tests were performed on groups of mice given 3.2 g/kg 25 vol% alcohol po, 30 min after administration of 10, 32, or 100 mg/kg of the natural, genuine valepotriate mixture po, and it was found that the valepotriates antagonized the alcohol effect. The effects on alcohol anesthesia (3.0 g/kg 20 vol% alcohol iv) of po administration of: chlordiazepoxide (0.1, 1.0, or 10 mg/kg), diazepam (0.1, 1.0, or 10 mg/kg), natural valepotriate mixture (10.0, 31.6, or 316.0 mg/kg), and the 3 individual components of the mixture—didrovaltratum (1.0, 31.6, 100.0, or 316.0 mg/kg), valtratum (31.6, 100.0, and 316.0 mg/kg), and acevaltratum (31.6, 100.0, and 316.0 mg/kg)—were also determined. Anesthesia was much prolonged by chlordiazepoxide and diazepam, and, to a lesser extent, by the 2 larger doses of the valepotriate mixture and acevaltratum. The 2 larger doses of valtratum shortened anesthesia, as did the 31.6 mg/kg dose of didrovaltratum. The effects of po administration of the above substances on alcohol toxicity were examined, and it was found that the valepotriate mixture, its components, and chlordiazepoxide did not increase alcohol toxicity, while larger diazepam doses lowered the alcohol LD₅₀.

365. Eickstedt, K. -W. von

DIE BEEINFLUSSUNG VON ALKOHOLWIRKUNGEN DURCH PHARMAKA IM TIERVERSUCH. [The influence of drugs on the effects of alcohol in animal experiments].

In: *Alkohol und Verkehrssicherheit*: Konferenzbericht der 5. Internationalen Konferenz über Alkohol und Verkehrssicherheit. [Alcohol and traffic safety: proceedings of the 5th International Conference on Alcohol and Traffic Safety]. Freiburg im Breisgau, West Germany, 1969. Freiburg im Breisgau: Hans Ferdinand Schulz Verlag, pp. I.123-I.126 (4 ref.) @ 1969.

G – exp. cont. – exp. comp. – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – CNS – sed., hypnot. – tranquilizers – *CAAAL-0

B-1010.

Experiments are described which investigated, in mice and rats, the combined effect of alcohol and drugs on motor coordination, prolongation of alcohol narcosis, and the relationship of the duration of alcohol narcosis to the blood alcohol concentration (BAC). Motor coordination was studied in rotarod tests, using mice treated with alcohol (31.6 g/kg po), chlordiazepoxide (CDP—31.6 mg/kg po), valmane (a new valerian derivative), or alcohol + 1 of the drugs. In the alcohol narcosis tests, also on mice, the effects of CDP (0.1, 1.0, and 10.0 mg/kg po), a natural valepotriate mixture (10.0, 31.6, and 316.0 mg/kg po), diazepam (0.1, 1.0, and 10.0 mg/kg po), and chlorpromazine (CPZ—0.001, 0.010, 0.100, and 1.000 mg/kg po) were determined. The relation of duration of alcohol

narcosis to the BAC was studied in male Sprague-Dawley rats given 2.65 g alcohol/kg iv, 2.65 g alcohol/kg iv 15 min before 0.88 g alcohol/kg iv, 0.316 mg CPZ/kg po + the larger alcohol dose, or the same CPZ dose + both alcohol doses. Lower doses of CDP and valmane antagonized the effect of alcohol on motor coordination, while higher CDP doses had an additive effect. All drugs tested significantly prolonged alcohol narcosis, the strongest effect being produced with CPZ. The duration of alcohol narcosis was found to be independent of the BAC. It is concluded that enhancement of the alcohol effect is caused by drug action on the CNS, and not on alcohol metabolism. Determination of the BAC cannot clarify the nature of an alcohol-drug interaction.

366. Eirich, R.

ÜBER DEN EINFLUSS VON ALKOHOL AUF ANTIBIOTIKA. [The influence of alcohol on antibiotics].

Dissertation, Medical Faculty of the University of Mainz, West Germany, 19 pp. (19 ref.),

1951.

G – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
– in vivo – in vitro – blood lev. – anti-infectants – *CAAAL-0 A-0660.

In vitro tests on the influence of alcoholic sol (1 cc ethanol in 20, 40, 60, and 80% sol) on the penicillin molecule (50,000 IU crystallized penicillin) disclosed that the 80% alcohol sol brought about inactivation of the drug after 1-2 hr. In vivo experiments on rabbits (injected im with 50,000 IU penicillin dissolved in distilled water and 5 cc 20% ethanol iv) disclosed that the penicillin blood level was somewhat increased. In tests with animals, alcohol increased somewhat and prolonged the blood level of streptomycin. The same results were obtained with ethanol and aureomycin given simultaneously po. Alcohol proved to be an excellent stabilizer in tests with terramycin.

367. Eis, G.

ALTDEUTSCHE HAUSMITTEL GEGEN TRUNKENHEIT UND TRUNKSUCHT. [Old German household remedies against drunkenness and alcoholism].

Med. Mschr. (Stuttgart), 15(4): 269-271 (15 ref.),

1961.

G – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – *CAAAL-9556-M3
A-0661.

Old German remedies against drunkenness are reviewed. The first warning voices against the praise of drunkenness appeared in the 16th century. Substances are discussed which were believed to prevent drunkenness, and others to be administered to the moderate drinker to make him intoxicated after little alcohol ingestion. The problem of inducing permanent abstinence in chronic alcoholics is also discussed.

368. Elbel, H.

TRUNKENHEITSBEGUTACHTUNG DURCH BLUTALKOHOLBESTIMMUNG:

DERZEITIGER STAND UNSERES WISSENS. [Diagnosis of intoxication by blood alcohol determination: current state of our knowledge].

Med. Welt (Stuttgart), 12: 1667-1671 (0 ref.),

1938.

G – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – blood lev. – absorp., distrib., stor. – metab. proc. – analg., antipyret. – stimulants – *CAAAL-195-U5
A-0662.

The current state of knowledge of diagnosis of intoxication by blood alcohol determination is discussed in some detail. Coffee does not influence the blood alcohol content, but has a marked detoxicating action which only lasts a few min. Smoking of only one cigarette markedly decreases efficiency of the intoxicated subject. Aspirin, pyramidon, and veronal have no antagonistic effect on blood alcohol; drunkenness is actually increased. Coramine seems to be detoxicating.

369. Elbel, H.

NEUES ZUR BLUTALKOHOLFRAGE (WIDMARK- ODER FRIEDEMANN-KLAAS-METHODE; ALKOHOLBESTIMMUNG IN FAULEM BLUT; WIRKUNG VON ASPIRIN, PYRAMIDON, VERONAL UND DEXTROENERGEN AUF DIE BLUTALKOHOLKURVE UND AUF DIE TRUNKENHEIT.) [New contribution on the blood alcohol question (Widmark or Friedemann-Klaas Test; determination of alcohol level in decaying blood; effect of aspirin, pyramidon, veronal and dextroenergen on the blood alcohol curve and on intoxication)].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 30: 218-226 (10 ref.), 1938.

G – exp. comp. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – analg., antipyret. – elect., water-bal. agents – *CAAAL-859-U5 A-0663.

In a comparative study, the Widmark Test proved to be more practical than the Friedemann-Klaas Test. Slightly better results in determining the alcohol level in decaying blood were reached with the Nicloux method. Aspirin, veronal, pyramidon, and dextroenergen had no effect on the blood alcohol level in human subjects.

370. Elbel, H.

NACHWEIS DER COFFEINWIRKUNG AUF BLUTALKOHOLGEHALT UND TRUNKENHEIT. [Demonstration of effects of caffeine on blood alcohol level and drunkenness]. Beitr. Gerichtl. Med. (Vienna), 15: 14-25 (18 ref.), 1939.

G – exp. cont. – DC (decrease) – mot. vehic. – humans – acute admin. – in vivo – dose resp. – blood lev. – mot. perform. – psychol. perform. – blood comp., sites, lymph, – cardiovasc. – CNS – stimulants – *CAAAL-0 A-1308.

2 subjects of the same age, weight, and build were tested for 6 weeks under various conditions of alcohol and caffeine consumption. Alcohol was administered in the form of beer (3.9% v/v), followed by caffeine in the form of coffee (.4 to .5 g caffeine/300 cc). To test performance, the subjects operated an apparatus which arbitrarily presented 1 of 12 stimuli, and the response in pressing the correct lever was timed (this was intended to simulate driving a truck). Blood samples were taken every 1/2 hr for the first 4 hr, then every hr. Subjective and objective observations on the feelings, moods, and sobriety of the subjects were also recorded. It is concluded that, although alcohol-increased reaction time is shortened by caffeine, the quality of the reaction is poorer and less effective; hence, contrary to being a sobering agent, caffeine intake can only lead to further impairment. Also, caffeine in large doses does not influence the blood alcohol level, and other experiments at Heidelberg University with therapeutic caffeine doses (0.1 and 0.2 g as Coca-Cola) have shown that small doses have no effect.

371. Elbel, H., and Schmelz, J.

ÜBER RAUSCHVERHÜTUNGSMITTEL. [Intoxication-preventing agents].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 33: 259-264 (6 ref.), 1941.

G – exp. cont. – exp. comp. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – mot. perform. – amphetamines – tranquilizers – *CAAAL-0 A-0664.

Two drugs, "Pekasin" and "Gothania antialcohol tablets", advertised as being capable of reducing the blood alcohol level and of neutralizing the performance impairment due to alcohol, were studied. Medical students ingested either 0.6, 0.8 and 1.0 g/kg of wine and 3 tablets of pekasin within 15 min, or 2.8 l of beer and 6 tablets of pekasin within 1 hr. Six gothania tablets—the same alcohol dosages were given—were ingested before the peak of the alcohol effect, and in another trial after the peak effect of alcohol. The results of the performance tests (putting curtain rings on a rod) showed no difference between the experimental and the control groups, thus proving the ineffectiveness of the two drugs.

372. Elbel, H.
PERVITIN UND ALKOHOL. [Pervitin and alcohol].
 Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 36: 90-100 (26 ref.), 1942.
 G – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – psychol. perform. – CNS – amphetamines – *CAAAL-4057-A1 A-0665.
- 2 volunteers received alcohol (2.8 l beer with 2.8% alcohol, and 100 cc gin with 36.8% alcohol) 8 times within 1 hr. On 4 occasions, nothing else was given. Twice the subjects received 9 mg pervitin before drinking; another 2 times, the same amount 1 hr after drinking. Tests were given in all 8 experiments. Pervitin given before drinking suppressed the effects of intoxication; given after drinking at the height of intoxication, it accelerated the return to normal performance.
373. Elbel, H.
BEDEUTUNG, NACHWEIS UND BEURTEILUNG DER ALKOHOLWIRKUNG IM VERKEHR (I) (II). [Significance, proof and evaluation of the effects of alcohol in traffic. (I) (II)].
 Med. Welt (Stuttgart), 20: 1106-1108, and 1151-1154 (13 ref.), 1951.
 G – exp. cont. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – CNS – indust. intox. – tranquilizers – *CAAAL-5982-J11 A-0666.
- Traffic accidents involving alcohol are discussed. Statistics based on blood alcohol determinations in 10,000 traffic accident cases are presented, showing different percentages of alcohol involvement on different days of the week, and different blood alcohol concentrations at different times of the day. The effects of “pekasin”, “gothania antialcohol tablets”, “bavarin 404”, and gasoline vapour were investigated. These influenced the personality picture, but not the blood alcohol level; fruit also failed to affect the concentration. A favourable effect of caffeine and pervitin on performance after alcohol was not demonstrated. Although these drugs at first seemed to have a sobering effect, judgement capacity was even less than with alcohol alone.
374. Elbel, H.
ÜBER ERNÜCHTERUNGSMITTEL FÜR KRAFTFAHRER. I. ALLGEMEINES ZUM PROBLEM DER ERNÜCHTERUNG. [A sobering-up drug for drivers. I. General considerations regarding the problem of sobering-up].
 Zentralblatt für Verkehrs-Medizin, Verkehrs-Psychologie und Angrenzende Gebiete (Alfeld/Leine), 1: 89-92 (0 ref.), 1955.
 G – general – DC (decrease) – DC (unchanged) – humans – absorp., distrib., stor. – metab. proc. – amphetamines – stimulants – tranquilizers – *CAAAL-0 A-0667.
- A general discussion is given on physiological and pharmacological investigations concerning alcohol and sobering agents. A number of remedies from folk-medicine are cited. It is pointed out that alcohol oxidation shows individual variations, being a function of the basic biochemical mechanism which may be disturbed by metabolic effects. Caffeine and pervitin, among others, are alleged to have an effect on inebriety without influencing alcohol metabolism. The author concludes that none of the so-called sobering agents (“pekasin”, “gothania antialcohol tablets”, “bavarin 404”, “alkstop”, “contra”, and “stop”) can influence a state of intoxication or alcohol metabolism after absorption.
375. Elbel, H., and Schleyer, F.
BLUTALKOHOL: DIE WISSENSCHAFTLICHEN GRUNDLAGEN DER BEURTEILUNG VON BLUTALKOHOLBEFUNDEN BEI STRASSENVERKEHRSDELIKTEN. [Blood alcohol: scientific foundations for the interpretation of blood alcohol findings in traffic violations].
 2nd ed. Stuttgart: Georg Thieme, 226 pp. (104-116, 163-166, 207-222) (887 ref.), 1956.

G – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – blood lev. – mot. perform. – psychol. perform. – amphetamines – analg., antipyret. – elect., water-bal. agents – hormones, hormone antag. – indust. intox. – nutritive agents – sed., hypnot. – stimulants – *CAAAL-8508-A1 A-0668.

Reviewed on the pages noted above is the literature on the influence of nicotine, caffeine, pervitin, levulose, insulin, vitamin B₆, analgetics, antipyretics, hypnotics (barbiturates in particular), dinitrophenol, dinitrocresol, and carbon dioxide on alcohol-induced performance impairment. Also reviewed is the literature on the relationship of various drugs, hormones, and vitamins with the alcohol metabolism, and their influence on it.

376. Elbel, H.

STEIGERUNG DER ALKOHOLWIRKUNG DURCH MEDIKAMENTE? [Drug enhancement of the effects of alcohol?].

Pharmazeutische Zeitung, Vereinigt mit Apotheker-Zeitung (Frankfurt a.M.), 107(45): 1542 (0 ref.), 1962.

G – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – cardiovasc. – metab. proc. – nutritive agents – stimulants – *CAAAL-0 A-0669.

The question whether drugs increase the effect of alcohol is asked in connection with the case of a man suffering from hypotonia, who consumed during a one-week period appreciable doses of anti-neuralgic drugs to combat grippe and fever. After alcohol ingestion, a blood alcohol level of 1.8°/oo was established. The man suffered a heart attack 1 hr after the blood sample was taken. The next day, he had no memory of the events of the day before. It is assumed that, in the case in question, the anti-neuralgic drugs caused a slowdown in the decomposition of ethanol and an accumulation of acetaldehyde. The amnesia could easily have been caused by the reported blood alcohol level of 1.8°/oo alone. The drugs could not have increased the blood alcohol level itself.

377. Elbel, H.

ZUR FRAGE DER KOMBINATIONSWIRKUNG VON ALKOHOL UND MEDIKAMENTEN. [The problem of the combined effects of alcohol and drugs].

Archiv für Kriminologie (Leipzig), 134: 83-86 (1 ref.), 1964.

G – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – psychol. perform. – antidepressants – *CAAAL-0 A-0670.

Attention and reaction ability under stress was tested in 6 human subjects—in sober condition, after 0.2 g of insidon, after alcohol (leading to a blood alcohol concentration of 0.8-1.2°/oo) and after the drug-alcohol combination. The alcohol was given 2 hr after the drug. The drug-alcohol combination had no effect on attention, but had a strong deteriorating effect on reaction performance.

378. Elzay, R. P.

LOCAL EFFECT OF ALCOHOL IN COMBINATION WITH DMBA ON HAMSTER CHEEK POUCH.

J. Dent. Res. (Chicago), 45(6): 1788-1795 (13 ref.), 1966.

E – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – metab. proc. – skel., muscle, skin – *CAAAL-12545-D2 B-0287.

Three groups of 20 male hamsters had cheek pouches painted with 7, 12-dimethylbenz-(a)-anthracene (DMBA, a carcinogen), at a concentration of 0.5% in heavy mineral oil (USP) or 50% ethyl alcohol by vol. The mortality rate with DMBA and alcohol was less, implying that this combination was less toxic. Tumefaction was more aggressive with DMBA-ethanol than with DMBA-oil treatment. Pouches receiving 1 dose of DMBA and ethanol 3 times/week displayed dyskeratosis (38%),

parabasilar budding (25%), and carcinoma in situ (13%), suggesting that ethanol acted locally as a promoting agent.

379. Erlanson, P., Fritz, H., Hagstam, K. -E., Liljenberg, B., Tryding, N., and Voigt, G.
SEVERE METHANOL INTOXICATION.
Acta Med. Scand. (Stockholm), 177(4): 393-408 (56 ref.), 1965.
E – general – case hist. – DC (antidotal) – post-mort. – humans – drug-dep. humans – blood lev. – other drug lev. – acid-base, blood pH, elect. – blood comp., sites, lymph – cardiovasc. – CNS – G.I. tract – liver, kidney – metab. proc. – respir. – senses – alcohols – elect., water-bal. agents – *CAAAL-0 B-0288.

4 patients with methanol poisoning were stuporous or comatose, and had severe acidosis. They were treated by haemodialysis, ethanol, and sodium bicarbonate; ethanol was administered according to Røe's principle. 3 died in the acute phase of the disease. The breakdown of methanol was virtually completely inhibited by ethanol in the survivor and 2 of the fatal cases; the other patient was not given sufficient alcohol to compensate for that removed by dialysis. Treatment advocated includes prompt administration of ethanol po or iv—about 50 g initially to a 70 kg patient, followed by 10-12 g/hr to maintain a blood ethanol concentration of about 1°/oo. The authors consider that the reason why the clinical value of ethanol treatment in methanol poisoning was questioned in earlier studies was that they were largely based on animal experiments with non-primates, the methanol metabolism of which is not the same as that of man.

380. Estable, J. J., Grezzi, J. W., and Varela Rodriguez, J.
TOXICIDAD COMPARADA DEL ALCOHOL Y LAS DISTINTAS BEBIDAS ALCOHÓLICAS DESTILADAS NACIONALES SOBRE PECES MANTENIDOS EN ACUARIOS: NOTA PREVIA. [Comparative toxicity of alcohol and of different distilled national alcoholic beverages on fish in aquariums: preliminary note].
Sociedad de Biología de Montevideo, Archivos (Montevideo), 21: 43-47 (0 ref.), 1954.
Sp – ES – FS – exp. comp. – congen. stud. – other org. – acute admin. – in vivo – dose resp. – *CAAAL-7548-A2 A-0671.

Fish were immersed in various concentrations of ethanol, cana, grappa, cognac, and whiskey, the final alcohol concentrations ranging between 15 and 35°/oo. The survival time varied between 3.7 and 39.5 hr, depending on the alcohol concentration, but varied little with the same concentrations of the different beverages. It is concluded that toxicity depends on the alcohol concentration, and not on the congeners in the beverages.

381. Estable, J. J., and Grezzi, J. W.
TOXICIDAD COMPARADA DEL ALCOHOL Y LAS DISTINTAS BEBIDAS DESTILADAS A.N.C.A.P. EN ANIMALES DE SANGRE CALIENTE. [Comparative toxicity of alcohol and of different A.N.C.A.P. distilled beverages on warmblooded animals].
Sociedad de Biología de Montevideo, Archivos (Montevideo), 21: 47-55 (9 ref.), 1954.
Sp – ES – FS – exp. comp. – congen. stud. – mammals – acute admin. – in vivo – dose resp. – blood lev. – respir. – *CAAAL-7548-A2 A-0672.

Lethal blood alcohol concentrations were determined in 10 dogs, 16 guinea pigs, and 73 mice after ip administration of alcohol, cana, grappa, cognac, and whiskey, all with a concentration of 40 g of absolute alcohol/100 cc. Death occurred at the same alcohol concentration, with an average value of 9.8 g/l of blood, and with extremes between 8.5 and 14.5 g. It was concluded that impurities (congeners) in the beverages (which contained impurity coefficients between 200 and 1,100) do not augment the toxicity; the ethyl alcohol content is the sole intoxicating factor.

382. Estable, J. J., Grezzi, J. W., and Varela Rodriguez, J.
ACCIÓN DE LA CONCENTRACIÓN DEL ALCOHOL Y LAS "IMPUREZAS" DE LAS BEBIDAS ALCOHÓLICAS DESTILADAS, SOBRE LA CURVA DE ALCOHOLEMIA EN EL PERRO. [Effect of concentration of alcohol and "impurities" in distilled alcoholic beverages on the blood alcohol curve in the dog].
 Sociedad de Biología de Montevideo, Archivos (Montevideo), 21: 56-63 (3 ref.), 1954.
 Sp - ES - FS - exp. comp. - congen. stud. - mammals - acute admin. - in vivo - blood lev. - other drug lev. - *CAAAL-7548-A2 A-0673.

5 fasted dogs received 1 g of absolute alcohol/kg po in concentrations between 10 and 40 g/100 cc. Each of the 5 dogs received, on different occasions, 48 hr apart: alcohol, cana, grappa, cognac, and whiskey. At concentrations of 10-40 g/100 cc, the blood alcohol level oscillated between 1.01 and 1.35 g/l; these levels fell gradually, disappearing completely 7-9 hr after ingestion. Analogous values were obtained with the urinary elimination 2-4 hr after the ingestion of the alcohol sol, the average values oscillating between 0.82 and 0.90 g/l. The authors conclude that the toxicity of the beverages is comparable to that of pure ethyl alcohol of equal concentration, and that the congeners have no toxic effect.

383. Estable, J. J., Grezzi, J. W., and Baraibar, E.
DETERMINACION CUANTITATIVA DEL COLAGENO Y LOS LIPIDOS HEPATICOS EN EL CURSO DE LA INTOXICACION CRONICA EXPERIMENTAL CON BEBIDAS ALCOHOLICAS DESTILADAS. [Quantitative determination of hepatic collagen and lipids in the course of experimental chronic intoxication with distilled alcoholic beverages].
 Facultad de Medicina de Montevideo, Anales (Montevideo), 44: 261-268 (19 ref.), 1959.
 Sp - ES - exp. cont. - exp. comp. - congen. stud. - mammals - chronic admin. - in vivo - liver, kidney - *CAAAL-8810-B2 A-1393.

40 albino rats, submitted to a diet rich in lipotropic factors and vitamins, were divided into 5 groups, and, for 15 months, the fluid intake consisted of a sol (10% alcohol concentration) of 1 of the following: alcohol sol, caña (local rum), "grappa", or brandy. Controls received only water. 1 animal of each group was periodically sacrificed; histological examinations were performed, and quantitative determinations made of liver collagen and fats. No significant increase of collagen was found in any group. The mean concentration of lipids was slightly higher in the treated groups than in the control group. There was a significant increase of liver fats in the caña and alcohol groups, and, to a lesser extent, in the grappa and brandy groups. Histological liver studies revealed normal parenchyma in all groups, with no marked alterations of lobulillar pattern, no cell infiltration, and no connective tissue proliferation. Treated animals showed a smaller weight gain, the difference with control values decreasing in the following order: brandy, alcohol, grappa, and caña. It is concluded that alcohol should be regarded as the main factor in liver alterations, and that congeners play a secondary, and relatively minor, role.

384. Estler, C. -J., and Ammon, H. P. T.
DER EINFLUSS DES ÄTHYLALKOHOLS UND EINES β -SYMPATHICOLYTICUMS AUF DEN EISENGEHALT VON LEBER, MILZ UND SERUM UND DIE EISENBINDUNGSKAPAZITÄT DES SERUMS. [The influence of ethyl alcohol and a β -sympatholytic agent on the iron content of liver, spleen, and serum, and on the iron-binding capacity of the serum].
 Arzneimittelforschung (Aulendorf), 17: 69-70 (21 ref.), 1967.
 G - ES - exp. comp. - DC (unchanged) - mammals - acute admin. - in vivo - dose resp. - blood lev. - blood comp., sites, lymph - liver, kidney - miscellaneous - *CAAAL-0 B-1011.

The effects of alcohol and a β -sympatholytic agent, kö 592 (1-isopropylamino-3 (m-toloxo-2-propanol HCl), alone and in combination, on the iron content of liver, spleen, and serum, and on the total

iron-binding capacity of serum, were investigated. Male Sprague-Dawley rats were given alcohol alone, kö 592 alone, or alcohol plus kö 592 (the first kö 592 injection was 50 mg/kg, and the following doses were all 25 mg/kg). The animals were sacrificed after 12 hr. Ethanol decreased serum iron content and serum iron-binding capacity. Kö 592 similarly reduced serum iron content and iron-binding capacity; however, the non-hemoglobin iron in liver and spleen was increased. In the combined dose, the individual effects of kö 592 predominated—2 hr after alcohol was given, the changes in iron content of liver and spleen were equal to those observed when kö 592 was given alone. Regardless of similar individual effects of alcohol and kö 592, the reduction of the serum iron content was not increased by their combined administration. The results do not allow the author to state reliably whether the effects of alcohol on iron metabolism are realized through the catecholamines.

385. Ettlinger, R., Fahlgren, H., and Reis, G. von
AKUTA INTOXIKATIONER MED MEPROBAMAT I KOMBINATION MED ALKOHOL.
 [Acute intoxication with meprobamate in combination with alcohol].
 Svenska Läkartidningen (Stockholm), 54: 2485-2490 (12 ref.), 1957.
 S – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – cardiovasc. – tranquilizers
 – *CAAAL-8963-E3 A-0674.

4 patients (1 woman), in whom overdosage of meprobamate in conjunction with moderate amounts of alcohol led to acute toxicosis, were admitted to a hospital in Stockholm. Caution is advocated in prescribing meprobamate to alcoholics.

386. Etzler, K., Joswig, E. H., and Mallach, H. J.
WEITERE ERGEBNISSE ÜBER DIE GEMEINSAME WIRKUNG VON ALKOHOL UND DIMETHYLSULFOXYD IM TIERVERSUCH. [Further results concerning the combined effect of alcohol and dimethylsulfoxide in animal trials].
 Arzneimittelforschung (Aulendorf), 16(9): 1228-1232 (9 ref.), 1966.
 G – ES – exp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin.
 – in vivo – metab. proc. – *CAAAL-0 B-0289.

Whereas in previous experiments the effects of alcohol (LD_{50}) had been diminished by small doses of dimethylsulfoxide (DMSO), when both substances were administered simultaneously, no decrease of the alcohol effect on white mice could be observed when the substances were given one after the other—whatever their order of succession. Small doses of DMSO even doubled or quadrupled the effects of large amounts of alcohol. The authors are unable to explain these effects on the basis of the animal experiments reported here, but tests on humans have led the authors to conjecture that the metabolites of the substance administered first may produce an increase of toxicity by influencing the original or metabolized form of the second substance.

387. Etzler, K., Hoenle, R., Joswig, E. H., Köhler, C., and Mallach, H. J.
PRÜFUNG DER STATISTISCHEN WECHSELWIRKUNG VON AETHYLALKOHOL UND DIMETHYLSULFOXYD IM TIERVERSUCH. [Investigation of the statistical interaction of ethyl alcohol and dimethylsulfoxide in animal experiments].
 Arzneimittelforschung (Aulendorf), 16: 1674-1680 (8 ref.), 1966.
 G – ES – exp. comp. – DC (unspec.) – mammals – acute admin. – in vivo – *CAAAL-0
 B-0290.

The combined effect of alcohol and dimethylsulfoxide (DMSO) was studied on 2940 survival times of white mice. These animals, in three equal groups received: (1) alcohol 1 hr before DMSO, (2) DMSO 1 hr before alcohol, or (3) alcohol and DMSO simultaneously. The mean survival time was, for (1), 616 min, for (2), 760 min, and for (3), 867 min. The mode of application (interval of application) had a statistically significant effect on survival time, on interaction of alcohol and DMSO,

and on alcohol and time factor. It could also be demonstrated that the single effects of alcohol, DMSO, and of the method of application were more important than all interactions.

388. Etzler, K.

TIEREXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE GEMEINSAME WIRKUNG VON AETHYLALKOHOL UND MOGADAN ROCHE. [Experiments on animals to investigate the combined effects of ethyl alcohol and mogadan].

Dissertation, Medical Faculty of Eberhard-Karls University, Tübingen, West Germany, 50 pp. (34 ref.), 1967.

G – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – sed., hypnot. – *CAAAL-0 B-0929.

The 24- and 72-hr LD₅₀'s of 50% and 100% ethanol and of mogadan were determined in albino mice, and, for a 24-hr period, were found to be 8.8, 9.0, and 5.5 g/kg, respectively. The behaviour of the animals was observed, and the results were tabulated. Using mortality and survival time as criteria, the combined effects of 100% ethanol and mogadan were studied in 980 mice (mean wt of 21.7 g), by po administration to groups of 20 animals of various decimal fractions of the 24-hr LD₅₀ of 1 substance, simultaneously with constant doses of the other; the fractions ranged from 0-100% of the LD₅₀'s. Controls received an equal mixture of water and linseed oil. Generally, as the variable dose of 1 substance increased, the mortality and survival time decreased, and it was concluded that this increased effect was due to a joint drug action. Behaviour of the animals was always influenced by the dominant substance. The hazards of combined intake of alcohol and mogadan in humans are stressed.

389. Etzler, K., Hoenle, R., Joswig, E. H., Köhler, C., and Mallach, H. J.

PRÜFUNG DER STATISTISCHEN WECHSELWIRKUNGEN ZWISCHEN AETHYLALKOHOL UND CARBAMAZEPIN, METHAQUALON UND NITRAZEPAM.

[Examination of the statistical interaction between ethyl alcohol and carbamazepine, methaqualone, and nitrazepam].

Arzneimittelforschung (Aulendorf), 19: 988-995 (16 ref.),

1969.

G – ES – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – respir. – anticonvulsants – sed., hypnot. – *CAAAL-0 B-0930.

The combined effects on survival time after po administration of methaqualone (M), nitrazepam (N), and carbamazepine (C) with alcohol po were investigated in 49 groups of mice (20 animals/group). The LD₅₀'s for ethanol, M, N, and C were found to be 9 g/kg, 650 mg/kg, 5500 mg/kg, and 1700 mg/kg, respectively. Various fractions of the LD₅₀'s of the test drugs and alcohol were combined, and dose-response curves were plotted. High doses of M with alcohol caused deep narcosis. Lower doses (260 mg/kg) of M with alcohol caused 100% mortality, while the same dose without alcohol only induced light sleep. Large doses of N plus alcohol induced sleep within 30 min, mucus secretions on the conjunctiva, and swelling of the eyes. With high doses of alcohol and low doses of N, the alcohol effect predominated. Low C doses plus high ethanol doses induced opisthotonic cramps. It is concluded that the effect of alcohol is increased by M and N, and decreased by C. The combined effect, however, is always less than additive.

390. Ewing, P. L.

ALCOHOL METABOLISM IN ARTIFICIAL FEVER.

Quart. J. Stud. Alcohol (New Haven), 1: 483-500 (28 ref.),

1940.

E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – cardiovasc. – respir. – indust. intox. – *CAAAL-111-A2 A-0675.

A study on dogs was made of the blood alcohol curve, the respiratory and urinary alcohol excretion, and the amount of alcohol oxidized at normal and increased (by diathermy or by administration of

dinitrophenol) body temperatures. Artificial fever induced by diathermy did not increase the rate of loss of alcohol administered iv to the dogs. 6 mg of 2,4-dinitrophenol sodium was administered after 2, 4, 6, and 8 cc 1:4 alcohol/kg. In several dogs receiving dinitrophenol after the 8 cc/kg alcohol dose, it was found that there was a more rapid fall in the blood alcohol compared to controls, although there was no increase in respiration (mechanically regulated). In 4 dogs which lived less than 2 hr after dinitrophenol, the alcohol loss was even more rapid. It is concluded that an increase in alcohol metabolism of about 20% takes place after dinitrophenol, and this must be related to the increased oxidation caused by the latter, and not to increased loss in respiration or increased temperature. The mechanism of this effect is not yet fully understood.

391. Fabre, R., Dérobert, L., and Le Guiner, G.
 ÉTUDE TOXICOLOGIQUE ET PHYSIOPATHOLOGIQUE DE QUELQUES ESSENCES. [A toxicological and physiological study of some essences].
 Institut National d'Hygiène, Recueil des Travaux (Paris), 2(2): 646-678 (29 ref.), 1946.
 F – exp. comp. – congen. stud. – mammals – acute admin. – in vivo – cardiovasc. – CNS – G.I. tract
 – liver, kidney – metab. proc. – miscellaneous – *CAAAL-4714-D2 A-1277.

The laws pertaining to various essences are reviewed, and the effects of the substances are described. 2 classes of essences are distinguished—the convulsion-producing (absinthe, hyssop, fennel, tansy, and sage essences) and the stupifying or motorically-exciting (anise, angelica, cassia, coriander, badiane, caraway, clove, balm-mint, and mint essences). Rats were fed these essences in both pure and diluted form. The animals were then sacrificed, their visceral organs examined histologically, and the amounts of essences in the organs were determined. In pure form, some of the essences were lethal—in particular: menthe, hyssop, lemon, amygdalus, and camomile. All the essences were found in the liver, brain, kidney, and adipose tissues; however, sometimes they were only in small quantities, indicating rapid elimination or oxidation within the organism. The major effect of the essences was acute hepato-nephritis. Intense hyperemia and blood-engorged vessels, especially around the kidneys, lungs, liver, and brain, were noted. The authors conclude that such essences taken through aperitifs can cause hepato-nephritic lesions, and that, though the dosages used on the rats do not correspond to what may be ingested by daily consumption of aperitifs, the essences have an indubitable toxicity.

392. Farrier, R. M., and Smith, R. H.
 CARBON TETRACHLORIDE NEPHROSIS: A FREQUENTLY UNDIAGNOSED CAUSE OF DEATH.
 J.A.M.A. (Chicago), 143(11): 965-967 (1 ref.), 1950.
 E – general – DC (add., infra-add., unspec. incr.) – humans – acid-base, blood pH, elect. – cardiovasc.
 – liver, kidney – respir. – anti-infectants – *CAAAL-5492-E7 A-0676.

Studies of 12 cases of carbon tetrachloride nephrosis showed that the condition is more common than present statistics reveal; it is usually wrongly diagnosed, since most cases are sporadic and non-industrial. Ingestion of alcohol proximal to the time of exposure to carbon tetrachloride greatly increases the danger of developing acute poisoning, lessens the chance for recovery, and increases the difficulty of obtaining an accurate history. The pathology and the treatment of carbon tetrachloride nephrosis is discussed.

393. Fazekas, J. F., Shea, J., and Rea, E.
 USE OF CHLORPROMAZINE IN THE MANAGEMENT OF ACUTE AND POSTALCOHOLIC STATES.
 International Record of Medicine (New York), 168: 333-339 (9 ref.), 1955.
 E – general – DC (unspec.) – drug-dep. humans – blood lev. – cardiovasc. – CNS – G.I. tract – liver,
 kidney – metab. proc. – nerv. syst. – senses – tranquilizers – *CAAAL-7368-N27 A-0677.

More than 500 alcoholics were treated with chlorpromazine for acute inebriation, alcoholic tremulousness, and delirium tremens. Acutely-intoxicated cases with 100 to 150 mg% blood alcohol received 50 mg chlorpromazine. This dosage produced sleep within 30 to 45 min. Additional doses were rarely necessary, but could be given without danger. Chlorpromazine neither affected the rate of disappearance of alcohol from the blood, nor increased the effect of alcohol by producing nervous system depression. It seemed to induce a fairly specific inhibition of subcortical areas.

394. Fazekas, J. F., Albert, S. N., and Alman, R. W.

INFLUENCE OF CHLORPROMAZINE AND ALCOHOL ON CEREBRAL HEMODYNAMICS AND METABOLISM.

Amer. J. Med. Sci. (Philadelphia), 230: 128-132 (11 ref.), 1955.

E – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – blood comp., sites, lymph – cardiovasc. – CNS – metab. proc. – tranquilizers – *CAAAL-7280-D1 A-0678.

21 convalescent hospital patients were divided into 3 groups. The first group of 8 subjects received 500 cc 10% ethanol iv, the second (6 subjects) received 200 or 300 mg chlorpromazine iv, and the third (6 subjects) received 1 liter 5% or 8% ethanol iv plus 100 mg chlorpromazine; 2 subjects in the last group were given chlorpromazine simultaneously with ethanol, and the remainder received the drug im after the ethanol. Clinically, it appeared that the degree of depression produced by the ethanol-chlorpromazine combination was only slightly greater than that produced by either drug alone. It is concluded that the 2 drugs in combination are neither pharmacologically synergistic nor quantitatively additive, but act on different sites in the CNS. An additive effect is conceivable, but only with doses of ethanol so large as to produce a dangerous degree of depression.

395. Fearn, H. J., and Hodges, J. R.

SYNERGIC EFFECTS OF AMYLOBARBITONE SODIUM AND ETHANOL.

J. Pharm. Pharmacol. (London), 5: 1041-1044 (8 ref.), 1953.

E – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – barbiturates – *CAAAL-6699-D2 A-0679.

2 types of experiments were conducted on white mice. The first test (300 mice) attempted to determine the effect of ethanol (50% v/v po) on the acute toxicity of amylobarbitone sodium (ABS) (20 mg/ml sol po); the LD₅₀ of each substance was established, and the amounts of ethanol required to produce 50% mortality in mice given 1/4, 1/2, or 3/4 of the LD₅₀ of ABS were determined. The second test (150 mice) investigated the anesthetic effects of both substances (ip) in combination; the median effective dose (MED)—the amount necessary to maintain anesthesia in 50% of the mice for 1 hr—of each substance was established, and the amounts of ethanol required to produce the median effective response in combination with 1/4, 1/2, or 3/4 of the MED of ABS were determined. Graph analysis of the results of both tests showed that the drug combination produced an additive synergism, but there was no evidence that ethanol potentiates the acute toxicity or anesthetic effect of ABS.

396. Feldmann, H.

ÜBER DEN EINFLUSS VON TRANQUILLIZERSUBSTANZEN AUS DER PHENOTHIAZINREIHE, AUS DER GRUPPE DER RAUWOLFIA-ALKALOIDE UND DES MEPROBAMAT AUF DIE ALKOHOLWIRKUNG BEIM MENSCHEN. [The influence of phenothiazine tranquilizers, the Rauwolfia alkaloid group and meprobamate on the effects of alcohol in man].

Dissertation, Medical Faculty of the University of Heidelberg, West Germany, 46 pp. (5 ref.), 1962.

G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – acute admin. – in vivo – mot. perform. – psychol. perform. – tranquilizers – *CAAAL-0

A-0680.

The following results were obtained in controlled tests with a number of tranquilizers combined with alcohol in healthy human subjects. 2 tablets of phasein-forte (reserpine and orphenadrine), and 1 g/kg alcohol showed no potentiation. Rivasin (reserpine) potentiated the effect of alcohol (1.5 g/kg)—performance was impaired to a considerable extent. 100 mg pacatal, a phenothiazine derivative, potentiated the effect of 1 g/kg alcohol; the potentiation was manifested by uninhibited behaviour. 5 tablets of salidyston (containing 5 mg imidazole derivative, 1 mg diethylaminoethylbenzylate and 20 mg barbiturate) and 1.3 g/kg alcohol showed synergism. 30 mg truxal, a thioxanthene derivative, showed the most appreciable potentiation of the alcohol effect (1.5 g/kg) in relation to the other substances.

397. Ferguson, J. K. W., Maharajh, M., and Warson, M. D.
EFFECT OF DERIVATIVES OF CYANAMIDE WITH AND WITHOUT ETHANOL ON BLEEDING WEIGHTS OF RATS AND MICE.
J. Pharmacol. Exp. Ther. (Baltimore), 113: 20-21 (0 ref.), 1955.
E – abst. – exp. comp. – DC (sensit.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – metab. proc. – unclass. ther. agents – *CAAAL-7350-B2 A-1278.

The effects of disulfiram and various cyanamide derivatives on the response to ethanol were studied in mice and rats. Blood levels of acetaldehyde and bleeding wt, expressed as % body wt, were determined as an index of altered response. When 0.75-1.5 ml/kg ethanol was administered a few hr after disulfiram or certain cyanamide compounds, the bleeding wt 1/2 hr later was reduced by 10-25%, and acetaldehyde levels were elevated. The sensitizing action of cyanamide compounds to ethanol was of shorter duration than that of disulfiram. Dicyandiamide and chloroformamidine hydrochloride increased acetaldehyde after ethanol, but did not affect bleeding wt. Cyanamide, monosodium cyanamide, urea complex with monosodium cyanamide, and calcium cyanamide, in doses of 0.06-0.24 g/kg, decreased bleeding wt and increased acetaldehyde levels. It is concluded that calcium cyanamide, with its advantages of freedom from sulphurous odour on the breath and more rapid control of the patient, is the most promising substance for therapy of alcoholics.

398. Ferrer Zanchi, A. G.
CONSTITUCION PARANOICA: ACCION DE LOS TOXICOS ALCOHOL-ACTEMIN-BARBITURICOS. [Paranoiac constitution: the effect of the alcohol-actemin-barbituric poisons].
Archivos de Medicina Legal (Buenos Aires), 20: 147-158 (37 ref.), 1950.
Sp – FS – general – conj. addict. – psychot. humans – psychol. perform. – amphetamines – barbiturates – sed., hypnot. – *CAAAL-5823-I1 A-0681.

The case is discussed of a 41 yr-old woman with paranoiac constitution who, from the effects of mental stress and the intense and prolonged use of alcohol, barbiturates (seconal and sedormid), actemin, benzedrine, and tobacco, exhibited "delirious ideas". Upon withdrawal of drugs and initiation of psychiatric treatment, the delirium and delirious ideas ceased. The paranoiac constitution remained, but this did not justify treatment.

399. Fiessinger, N., Bénard, H., Courtial, J., and Dermer, L.
COMBUSTION DE L'ALCOOL ÉTHYLIQUE AU COURS DE LA PERFUSION DU FOIE. [Combustion of ethyl alcohol in the course of circulation of the liver].
C.R. Soc. Biol. (Paris), 122: 1255-1258 (0 ref.), 1936.
F – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – in vitro – blood lev. – liver, kidney – metab. proc. – unclass. ther. agents – *CAAAL-2811-A2 A-0682.

Combustion of ethanol was studied in the isolated dog liver. The quantity of ethanol (1.5-2 g/l) in blood diminished regularly with time in a linear relationship. The total amount of alcohol was irrelevant. Potassium cyanide (0.20-0.30 g/l), and, to a lesser extent, potassium hyposulfite (1-2 g/l), retarded the oxidation of alcohol. 2,4-dinitrophenol (20 mg/l) had no effect.

400. Figueroa, R. B., and Klotz, A. P.
 THE EFFECT OF WHISKEY AND LOW-PROTEIN DIETS ON HEPATIC ENZYMES IN RATS.
 Amer. J. Dig. Dis. (New York), nsv. 9(2): 121-127 (14 ref.), 1964.
 E – exp. cont. – congen. stud. – mammals – chronic admin. – in vivo – liver, kidney –
 *CAAAL-10689-B2 A-1394.

A control group (1) of female rats received regular chow and water ad libitum; group 2 received regular chow plus 5 cc doses of 33% ethanol by stomach tube 5 days/week; group 3 received regular chow and a 50% sol of whiskey plus 5 cc 83% whiskey (equal to 33% pure ethanol) 5 days/week by stomach tube; group 4 received a low-protein diet of 50% chow and 50% sucrose (total protein content of 12%); group 5 received low protein as in group 4 and ethanol as in group 2; and group 6 received low protein as in group 4 and whiskey as in group 3. The rats were killed after 6 weeks, and the livers were examined. Some fatty changes, accentuated by the low protein diet, were found in the ethanol and whiskey groups. In contrast to ethanol, whiskey induced a significant decrease in alcohol dehydrogenase. Isocitric dehydrogenase was decreased in group 3, and, in group 2, glutamic transaminase was decreased and glutamic oxaloacetic transaminase increased. It is suggested that whiskey may contain a factor which potentiates the deleterious effects of alcohol in intrahepatic enzymes.

401. Filomusi-Guelfi, G.
 SULLA RAPIDITÀ E INTENSITÀ DELL'AVVELENAMENTO PER ACIDO ARSENIOSO SOMMINISTRATO NEL CAFFÈ O NEL CAFFÈ CON ALCOOL. [The rate and degree of poisoning by arsenic acid administered in coffee or in coffee with alcohol].
 Annali Universali di Medicina e Chirurgia (Milan), 281: 401-430 (13 ref.), 1887.
 I – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – species or sex diff. – G.I. tract – unclass. ther. agents. – *CAAAL-0 A-0683.

In a series of controlled experiments, increasing doses of arsenic acid (10 cg-1.60 g) in coffee or water as vehicles were administered to 2 dogs (designated as "A" and "B") in the presence and absence of alcohol. The arsenic administered to A was dissolved in 5 cc alcohol prior to infusion in coffee. In the case of B, the arsenic was added to a sol of coffee and alcohol (5 cc). The toxic action was greater in A, which died after a 60 cg dose, despite the fact that it was bigger than B, which died after 1.60 g arsenic. In the presence of 20 cc alcohol, the toxic action receded (attributed to emetic stimulation). The mechanism of this interaction is not elucidated, but it is conjectured that individual resistance plays a major part. No marked differences were noted between coffee and water, with regard to emetic action.

402. Finer, M. J.
 HABITUATION TO CHLORDIAZEPOXIDE IN AN ALCOHOLIC POPULATION.
 J.A.M.A. (Chicago), 213(8): 1342 (0 ref.), 1970.
 E – general – conj. addict. – drug-dep. humans – tranquilizers – *CAAAL-0 B-0931.

The hazard of the present practice of generous prescription of chlordiazepoxide for relief of anxiety and tension in alcoholics has become apparent during an alcoholism treatment program which has thus far involved 630 alcoholic patients who volunteered to remain in a special ward for 6 weeks. The frequent and intense pleading of these patients, intoxicated or sober, for chlordiazepoxide is so notable that habituation is easily revealed. Patients for whom chlordiazepoxide is prescribed to subdue alcohol withdrawal symptoms may experience a chlordiazepoxide abstinence syndrome when deprived of chlordiazepoxide as long as 4 weeks after sobriety. Chlordiazepoxide habituation can be expected to increase the "hazardous potential sequelae" to alcohol intoxication, including: comatose states, accidental injuries, aspiration of vomitus, pneumonia, neglect of other illnesses, and even death. Compara-

tive experiences of the author have shown that diazepam is far less habituating than chlordiazepoxide, but is nevertheless equally as effective for control of alcohol withdrawal.

403. Finkle, B. S., Biasotti, A. A., and Bradford, L. W.
 THE OCCURRENCE OF SOME DRUGS AND TOXIC AGENTS ENCOUNTERED IN
 DRINKING DRIVER INVESTIGATIONS.
 J. Forensic Sci. (Mundelein), 13(2): 236-245 (2 ref.), 1968.
 E – stat. surv. – conj. addict. – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans
 – blood lev. – mot. perform. – analg., antipyret. – autocoids – coagulants – hormones, hormone antag.
 – sed., hypnot. – *CAAAL-12654-S7 B-0291.

This survey is an evaluation of the incidence and significance of drugs encountered in 3,409 routine drinking driver cases in the County of Santa Clara, California, during 1966. 21% of the cases indicated concurrent drug use; 107 different specific drugs were named under routine questioning. These drugs are tabulated in 20 groups according to their physiological action. Ataractics and ataxics accounted for 19.3% of occurrences, sedatives and hypnotics for 7.5%, and analgesic narcotics for 3.8%. In the fraction of cases in which the blood alcohol concentrations were less than 0.15% w/v, and the subjects exhibited definite symptoms of intoxication, 21% were found through screening tests to have a significant concentration of other drugs in the blood sample submitted for alcohol analysis.

404. Finkle, B. S.
 DRUGS IN DRINKING DRIVERS: A STUDY OF 2,500 CASES.
 Journal of Safety Research (Chicago), 1(4): 179-183 (11 ref.), 1969.
 E – stat. surv. – DC (unspec.) – med.-leg. – mot. vehic. – humans – species or sex diff. – amphetamines
 – analeptics – analg., antipyret. – antidepressants – antispasmodics – barbiturates – sed., hypnot. –
 stimulants – tranquilizers – *CAAAL-0 B-0932.

During the years 1966-1968, a statistical study was made by the Laboratory of Criminalistics of Santa Clara County, California, based on chemical testing of urine and blood samples from 10,436 persons arrested during routine drinking driver investigations. 2,559 cases, involving 2,688 drug occurrences and 273 different drugs, were studied. 13% (1,406 cases) involved agents legally defined in California as drugs unfit for self-medication, and requiring a doctor's prescription. In addition to results from requests by police for alcohol and/or drug analyses, analytical data was compiled from the fraction of cases in which the blood alcohol concentration (BAL) was less than 0.15% w/v, and in which the subjects exhibited overt symptoms of intoxication normally associated with a BAL in excess of 0.24%. 700 drug analyses were performed, of which 22% were positive for 1 or more of 24 drugs; with the exception of salicylate and caffeine, every analysis involved a dangerous, problem drug. A distribution by age and sex of these individuals in which drugs were detected is made, and the administrative usefulness and judicial outcome of the cases is noted, possibly to be used as a guideline in selecting future cases for analysis.

405. Fischer, E.
 THE COUNTERACTION OF WEAK CONCENTRATIONS OF AZIDE ON THE
 DEPRESSING EFFECT OF ALCOHOL ON THE OXYGEN CONSUMPTION OF BRAIN
 SLICES.
 In: Himwich, Harold E., ed. *Alcoholism: Basic Aspects and Treatment*. Washington: American
 Association for the Advancement of Science, Pub. No. 47, pp. 19-27 (12 ref.), 1957.
 E – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – in vitro – dose
 resp. – absorp., distrib., stor. – CNS – metab. proc. – *CAAAL-0 A-0684.

Experiments on rat brain slices showed that, although alcohol and azide, added simultaneously, acted additively to a certain extent on the activity and resting respiration, azide between 10^{-5} and 10^{-6} M

almost completely counteracted the depressing effect of 1.2% alcohol. When alcohol was added up to 1 hr after the azide, inhibition of excess respiration was almost equally suppressed, although azide added after alcohol showed steadily weakened power of counteraction as the time interval between administration of the two was increased.

406. Fischer, H. -D., and Oelssner, W.

DER EINFLUSS VON HEXOBARBITAL UND PHENOBARBITAL AUF DIE ALKOHOLELIMINATION BEI KANINCHEN. [The influence of hexobarbital and phenobarbital on the alcohol elimination in rabbits].

Med. Exp. (Basel), 3: 213-218 (24 ref.),

1960.

G – ES – FS – exp. cont. – exp. comp. – DC (unspec.) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – barbiturates – *CAAAL-9790-A2 A-0685.

The alcohol elimination rate from the blood of 10 rabbits was established with and without barbiturate pretreatment. Hexobarbital (50 mg/kg, im) was given to 7 rabbits for 1-5 days, and phenobarbital (40-80 mg/kg, im) was given to 3 rabbits for 3-4 days. The alcohol elimination rate after pretreatment varied between 163 and 286 mg/kg per hr, and the increases over the control rate were 20-43% for hexobarbital and 12-23% for phenobarbital. Maximum effect was obtained after 4-5 days pretreatment.

407. Fischer, H. -D., and Oelssner, W.

DER EINFLUSS VON BARBITURATEN AUF DIE ALKOHOLELIMINATION BEI MÄUSEN. [The effect of barbiturates on alcohol elimination in mice].

Klin. Wschr. (Berlin), 39(23): 1265 (5 ref.),

1961.

G – exp. cont. – exp. comp. – DC (unspec.) – mammals – acute admin. – in vivo – metab. proc. – barbiturates – *CAAAL-10084-A2 A-0686.

The alcohol elimination rate in mice was determined with and without pretreatment with hexobarbital and phenobarbital. 50 mice, of which half had been pretreated with 50 mg/kg ip of either barbiturate for 5 days, received 1.5 g/kg ethanol ip. Every 30 min, 5 mice from each group were killed and homogenized. Alcohol was determined by the Bucher and Redetzki method, and the elimination rate calculated. It was found that hexobarbital increased alcohol elimination by 20-25%; phenobarbital was somewhat less effective.

408. Fischer, H. -D.

DER EINFLUSS VON BARBITURATEN AUF DIE ENTGIFTUNGSGESCHWINDIGKEIT DES ÄTHANOLS. [The influence of barbiturates on the rate of detoxication of ethyl alcohol].

Biochem. Pharmacol. (London), 11: 307-314 (32 ref.),

1962.

G – ES – exp. cont. – DC (unspec.) – mammals – acute admin. – in vivo – metab. proc. – barbiturates – *CAAAL-10319-A2 A-0687.

8 male rabbits were given hexobarbital (50 mg/kg im) for 5 days and then killed; controls received alcohol 20 hr prior to the test. Liver wt was increased to 25.9 g/kg, compared to 20.5 in controls. Liver alcohol dehydrogenase content was 1.53 mg/g of liver, and 1.82 mg/g in controls. No difference between treated rabbits and controls in terms of body wt was observed in the amount of alcohol dehydrogenase found in the rabbit bodies. The ratio of diphosphopyridine nucleotide (DPN) to its oxidized form (DPNH) was increased to 2.35, compared to 1.77 in controls. The alcohol elimination rate was determined after injections of 1 g/kg 33% alcohol iv. A positive correlation was observed between the alcohol elimination rate and the DPN:DPNH ratio; the increase in this ratio in treated animals is of the same magnitude as the increased alcohol elimination found in hexobarbital-pretreated rabbits in a previous experiment (Fischer, H. -D., and Oelssner, W., Med. Exp. (Basel), 3: 213-218, 1960).

409. Fischer, H. -D.
ZUR ALKOHOLELIMINATION BEI KANINCHEN NACH CCL₄-VERGIFTUNG. [On the elimination of alcohol in rabbits after CCl₄ poisoning].
Medicina et Pharmacologia Experimentalis (Basel), 17(1): 60-64 (17 ref.), 1967.
G – ES – exp. cont. – DC (unspec.) – mammals – acute admin. – in vivo – blood lev. – liver, kidney – metab. proc. – anti-infectants – *CAAAL-0 B-0292.

Rabbits received 20% ethanol iv, and then 2.5 ml/kg CCl₄ po. It was found that, in the early stage of acute CCl₄ poisoning, the alcohol elimination was unchanged, but, after 8 hr, a markedly increased rate of elimination was observed which persisted up to the 14th hr. After this time, the elimination rate decreased rapidly to an abnormally low level, and death soon occurred—1/2 of the animals died after 15-22 hr. The results support the hypothesis that the liver NAD:NADH ratio is of consequence to alcohol elimination.

410. Fischer, I.
SÄREGEN SVAMPFÖRGIFTNING. [Unusual mushroom poisoning].
Svenska Läkartidningen (Stockholm), 42: 2513-2515 (1 ref.), 1945.
S – general – case hist. – DC (sensit.) – humans – cardiovasc. – CNS – G.I. tract – miscellaneous – *CAAAL-0 A-1249.

An occurrence of *Coprinus atramentarius* mushroom poisoning in 3 adults is described, in which symptoms, which are sometimes seen after an overdose of amyl nitrite, commenced 10 min after alcohol ingestion, lasted 2 hr, and disappeared completely after 5 hr. They consisted of heart palpitation, vertigo, general malaise, intense headache, anxiety, dry mouth, ataxia, hyperanemia, and nausea and vomiting. A fourth adult in the group, a domestic, ate exactly the same meal as the others, but without the alcohol; she experienced no reaction whatsoever. Such symptoms are subject to recurrence after a few days if alcohol is consumed, even without further mushroom consumption. Possible mechanisms are alcohol-accelerated absorption, anaphylaxis, or accidental inhalation of calcium cyanamide, a common fertilizer. Treatment is not mandatory, as the symptoms are neither dangerous nor of long duration; however, calcium chloride or glucomate injections may be given to alleviate discomfort.

411. Fisher, R. S., Walker, J. T., and Plummer, C. W.
QUANTITATIVE ESTIMATION OF BARBITURATES IN BLOOD BY ULTRA-VIOLET SPECTROPHOTOMETRY. II. EXPERIMENTAL AND CLINICAL RESULTS.
Amer. J. Clin. Path. (Baltimore), 18: 462-469 (4 ref.), 1948.
E – stat. surv. – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – blood lev. – sed., hypnot. – *CAAAL-0 A-0688.

Experiments were conducted on dogs and human subjects to investigate the concentration of barbiturate in the blood, with respect to drug type, dosage, and lapse of time after ingestion. 10 fatal cases of barbiturate poisoning are presented, 4 of which showed concomitant alcohol use; the latter group all had barbiturate levels of 2.5 mg/100 ml or less (pentobarbital or butethal) and blood alcohol levels of 0.13-0.37%. In 1 case, death resulted only 1 1/2 hr after ingestion of 0.8 g butethal and some whiskey (blood alcohol—0.13%). That death occurred from combinations of alcohol and barbiturates in concentrations which would not ordinarily be considered fatal, should focus attention on the very real danger of indiscriminate prescribing of barbiturate sedation for alcoholics attempting to "taper off".

412. Fiske, H. M.
ARSENIC AS ANTIDOTAL TO ACUTE ALCOHOLISM.
Pacific Medical and Surgical Journal (San Francisco), 16: 592-593 (1 ref.), 1875.

E – general – case hist. – DC (antidotal) – drug-dep. humans – unclass. ther. agents – *CAAAL-0 A-0689.

The author recounts the strange case of an alcoholic medical colleague given to drinking sprees of 1-3 weeks duration. When at any time he wanted to sober up, he would drink 1/2 oz of Fowler's arsenic sol, repeating the dose in 1 hr and at subsequent intervals if necessary, until sometimes he had swallowed 5-6 oz. "He would then be apparently as fresh and vigorous as though he had never been intoxicated, from two to five hours being all the time generally necessary to bring him to his normal condition." The author adds that, "Often have I been sent for to consult with him and found him unfit for business, when he would retire to a room, call for or take from his saddle bags his 'Fowler,' as he called it, and in two or three hours he would be ready for business."

413. Fiske, J. P.
SOME PECULIAR EFFECTS FROM LARGE DOSES OF HYOSCINE IN A CASE OF ACUTE ALCOHOLISM.

Medical Record (New York), 44: 142 (0 ref.), 1893.

E – general – case hist. – DC (unchanged) – humans – mot. perform. – CNS – analg., antipyret. – autonomic agents – *CAAAL-0 A-0690.

The author recounts the case of a 40 yr-old man suffering from insomnia and tension who had been drinking constantly for some days and nights. His speech was articulate, but he was very nervous and restless, and at times he conversed with hallucinations of his friends. He was given 2 injections of 1/100 grains hyoscine sc, 45 min apart, then 1/50 grains 9 hr later. The effects of the third dose were motor paralysis (speech, senses, and gait) and some circulatory disturbances. After 2 1/2 hr, the effects disappeared, and 1/2 grain morphine was administered. Little improvement was shown, and a second dose of 1/50 grains hyoscine was given 6 hr later, with the same effects as before. Chloral and bromides were then administered in large doses, followed by atropine and strychnine sc, with very favourable results. At no time did the hyoscine produce a hypnotic effect and the author concludes that the purported value of hyoscine in alcoholic mania and insomnia of acute alcoholism is much overrated.

414. FitzGerald, M. G., Gaddie, R., Malins, J. M., and O'Sullivan, D. J.
ALCOHOL SENSITIVITY IN DIABETICS RECEIVING CHLORPROPAMIDE.

Diabetes (New York), 11(1): 40-43 (26 ref.), 1962.

E – exp. comp. – DC (sensit.) – humans – acute admin. – in vivo – blood lev. – other drug lev. – cardiovasc. – metab. proc. – respir. – hormones, hormone antag. – *CAAAL-9732-E3 A-1279.

The incidence of alcohol sensitivity in 100 diabetics receiving chlorpropamide is reported. The sensitivity reaction consists invariably of flushing, which may be accompanied by conjunctival injection, a pounding headache, and a feeling of breathlessness. It begins 3-10 min after ingestion of even a small amount of ethyl alcohol, reaches a maximum intensity in about 20 min, and usually lasts about an hr. Most of the diabetics were taking 350 mg chlorpropamide/day. Of the 65 who acknowledged taking alcohol since treatment began, 22 had experienced at least mild symptoms of intolerance. In a series of experiments, it was found that, after administration of 25 ml of alcohol to diabetics receiving chlorpropamide, there was no observable increase in blood acetaldehyde levels, nor in the urinary concentration of 5-hydroxyindolyl acetic acid. It is concluded that, although the alcohol-chlorpropamide reaction closely resembles that seen following alcohol in patients receiving tetraethylthiuram disulfide, cyanamide, and several other substances, a similar mechanism of action is not observed.

415. Flamm, S.
KOMBINATORISCHE WIRKUNGEN VON COFFEIN UND ALKOHOL. [Combined effects of caffeine and alcohol].

Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 143: 79-87 (19 ref.), 1929.
 G – exp. cont. – DC (decrease) – mammals – in vitro – CNS – metab. proc. – nerv. syst. – stimulants
 – *CAAAL-0 A-0691.

In experiments on caffeine antagonism, the excitatory effect of caffeine on the isolated peripheral nerve and the degree to which caffeine is capable of antagonizing the depressant effect of alcohol were studied. Results showed that: 1) alcohol in non-narcotic doses effected a prolonged and constant increase of chronaxy (excitation depression) in the peripheral motor nerve; 2) a 0.05% caffeine sol effected a prolonged rise in excitation, and, after a bath in a Ringer sol, an irreversible decrease in excitation remained; and 3) in combination tests, a 0.05% caffeine sol was found to antagonize the alcohol effect (the effect depending on the depth of the narcosis). The possibility of different sites of action of the excitatory and antiexcitatory factors is discussed.

416. Fleming, P.
 DRUGS AND ACCIDENTAL DEATH: SOME POSSIBLE CAUSES OF SO-CALLED UNEXPLAINED SUDDEN DEATHS.
 J. Kansas Med. Soc. (Topeka), 66(1): 33-35 (10 ref.), 1965.
 E – SEC – general – DC (add., infra-add., unspec. incr.) – drug-dep. humans – nerv. syst. – sed., hypnot. – tranquilizers – *CAAAL-0 B-0157.

The possible effect of drug interaction causing potentiation is described with respect to sudden unexplained deaths. It is pointed out that the tranquilizers may potentiate certain anesthetics, barbiturates, narcotics, alcohol, antihistamines, sedatives, and hypnotics, and that the effect may be so great that the properties of a drug affected may be quadrupled. This was demonstrated in the case of a 50 yr-old man with a drinking problem who was found dead. Careful inquiries revealed that death was due to the combined action of 1 tranquilizing pill, a 2 oz quantity of Scotch, and 1 capsule of a sedative for sleep.

417. Fleming, R., and Reynolds, D.
 EXPERIMENTAL STUDIES IN ALCOHOLISM. IV. ATTEMPTS TO MODIFY THE CONCENTRATION OF ALCOHOL IN THE BLOOD AFTER INTRAVENOUS ADMINISTRATION OF ALCOHOL.
 J. Pharmacol. Exp. Ther. (Baltimore), 54: 236-245 (15 ref.), 1935.
 E – exp. cont. – exp. comp. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – G.I. tract – liver, kidney – metab. proc. – respir. – anticonvulsants – autonomic agents – hormones, hormone antag. – *CAAAL-1110-A1 A-0692.

The influence of various substances, and of diathermy upon the alcohol concentration in human blood was tested in 20 humans, most of them heavy drinkers; 0.6 cc absolute alcohol/kg as 20% sol was injected iv. The elevated body temperature by diathermy apparently increased the rate of disappearance of alcohol from the blood. The various substances such as milk, olive oil, magnesium sulfate, insulin, and epinephrine hydrochloride, had no significant influence on the rate of disappearance. Some of the implications of these findings are pointed out.

418. Foerster, R.
 UEBER DIE WIRKUNG DES METHYLALKOHOLS. [The action of methyl alcohol].
 Munchen. Med. Wschr. (Munich), 59(5): 248-251 (38 ref.), 1912.
 G – SEC – general – review – DC (decrease) – humans – alcohols – *CAAAL-0 A-0693.

The author outlines his theory that methanol does not in itself exercise a toxic influence, and that methanol poisonings are the result of an allergic reaction in certain sensitive humans. One conclusion,

based on his theory, is that the addition of ethanol to methanol does not make it toxic; this fact is substantiated by the observation that ethanol-methanol mixtures are often consumed without causing the appearance of symptoms of poisoning. That ethanol-methanol mixtures (at least in combinations of certain proportions) are often drunk without harmful results was a highly significant observation later followed by Røe (*Acta Med. Scand. (Stockholm)*, 113(6): 558-608, 1943) and others, although further research has not borne out the author's conclusion, and his theory is not now accepted.

419. Forbes, J. C., and Duncan, G. M.

EFFECT OF AMPHENONE ON THE ADRENAL RESPONSE OF RATS TO ALCOHOL INTOXICATION.

Quart. J. Stud. Alcohol (New Haven), 20: 5-12 (27 ref.), 1959.

E – exp. cont. – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – blood lev. – glands – *CAAAL-8440-A2 A-0694.

After receiving 0.5% of amphenone (1,1-bis(para-aminophenyl)-1-methyl propanone) in their diet for 7-14 days, male rats were given 3 g/kg 10% alcohol sol. The animals were sacrificed 1/2, 1, 4, and 6 hr after alcohol. The amphenone had no effect on intensity or duration of intoxication, nor did it influence the rate of alcohol intoxication as measured by the average drop from the 1 hr period to the end of the experiment. Average reduction of the blood alcohol was 36 mg/100 g blood/hr in controls and in amphenone groups. The rate of disappearance was not linear, but was consistently greater during the first 3 hr than during the last 2 hr.

420. Forney, R. B., and Hulpieu, H. R.

LACK OF EFFECT OF CARBUTAMIDE (BZ-55) ON THE METABOLISM OF ALCOHOL.

Diabetes (New York), 6(1): 28-30 (8 ref.), 1957.

E – exp. cont. – DC (unchanged) – mammals – chronic admin. – in vivo – blood lev. – metab. proc. – hormones, hormone antag. – *CAAAL-8375-A2 A-1280.

The effect of carbutamide on the 3 stages of ethanol metabolism was studied in 6 dogs. Test doses of alcohol (1 g/kg) were injected iv before, during, and after po carbutamide administration. Blood samples were collected at 45 min, and at 2, 3, 4, 5, and 6 hr after the ethanol infusion. The rate of disappearance of alcohol from the blood, and changes in blood acetaldehyde, blood pyruvate, and blood glucose were determined. It was found that the curves representing the rate of ethanol metabolism (stage 1) and the increases in blood acetaldehyde after alcohol administration (stage 2) were essentially the same with and without carbutamide medication. Blood pyruvate levels (stage 3) were lower during carbutamide treatment than during control periods, but were still within the normal range. The 20 mg% decrease in blood glucose observed during carbutamide treatment was not correlated with alcohol administration. It is concluded that daily dosage po with carbutamide for as long as 2 weeks has no effect on ethanol metabolism.

421. Forney, R. B., Hulpieu, H. R., and Hughes, F. W.

THE COMPARATIVE ENHANCEMENT OF THE DEPRESSANT ACTION OF ALCOHOL BY EIGHT REPRESENTATIVE ATARACTIC AND ANALGESIC DRUGS.

Experientia (Basel), 18(10): 468-470 (10 ref.), 1962.

E – GS – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – CNS – analg., antipyret. – tranquilizers – *CAAAL-10140-D2 A-0695.

A method is described to measure the potentiating action of tranquilizers and analgetics on alcohol depression. The immobility of mice was the criterion of depression. Five tranquilizers (reserpine, 0.125 mg/kg; chlorpromazine, 1.5 mg/kg; phenaglycodol, 10 mg/kg; meprobamate, 13.7 mg/kg; and hydroxyzine, 24 mg/kg) potentiated the effect of alcohol in various degrees. Of three analgetics (morphine, codeine, and d-propoxyphene), only morphine potentiated alcohol, and this was in a dosage of 100 mg/kg.

422. Forney, R. B., and Hughes, F. W.
PERFORMANCE DATA OBTAINED BY ADDITION OF TRANQUILIZERS WITH ALCOHOL IN HUMAN SUBJECTS.
 Third International Meeting in Forensic Immunology, Medicine, Pathology and Toxicology, Plenary Session II: "Drugs and Driving". London, England, 8 pp. (0 ref.), 1963.
 E – exp. – presentation – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – CNS – senses – antidepressants – stimulants – *CAAAL-0 A-0696.

The effects of alcohol, tranquilizers, and their combination on human performance were studied. Delayed auditory feedback was used in connection with 9 reading, arithmetical, and colour (modification of the Stroop test) tests. Caffeine (500 g) did not alter the behavioural spectrum, and was ineffective in antagonizing alcohol. In some of the tests, alcohol and caffeine combinations caused greater impairment than can be calculated from the individual actions of the drugs. A more definitive alcohol antagonism was evident with the use of a true anti-depressant, such as nortriptyline.

423. Forney, R. B., Hughes, F. W., and Hulpieu, H. R.
POTENTIATION OF ETHANOL-INDUCED DEPRESSION IN DOGS BY REPRESENTATIVE ATARACTIC AND ANALGESIC DRUGS.
 Quart. J. Stud. Alcohol (New Haven), 24(1): 1-8 (8 ref.), 1963.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – mot. perform. – CNS – analg., antipyret. – tranquilizers – *CAAAL-10828-D2 A-0697.

The potentiating action of some tranquilizing and analgesic drugs on the effects of ethanol was investigated in dogs. 3 to 5 dogs were used for each dose of the drugs. 1 g/kg of ethanol was used as the standard dose. A true potentiation of the effect of alcohol was demonstrated for meprobamate (30 mg/kg), phenaglycodol (30 mg/kg), chlorpromazine (1 mg/kg), and reserpine (0.032 mg/kg). The excitatory state induced by hydroxyzine (30 mg/kg) was antagonized by ethanol. The effects of morphine (0.16 mg/kg) and d-propoxyphene (2.5 mg/kg) were also potentiated, but not that of codeine (2.5 mg/kg).

424. Forney, R. B., and Hughes, F. W.
BEHAVIORAL EFFECTS ON THE RAT OF BENZQUINAMIDE AND BENZQUINAMIDE-ALCOHOL COMBINATIONS.
 Arch. Int. Pharmacodyn. (Gand), 142(1-2): 237-242 (6 ref.), 1963.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – psychol. perform. – CNS – tranquilizers – *CAAAL-10625-J2 A-0698.

Rats were trained to avoid a shocking stimulus. Benzquinamide (10, 20, and 30 mg/kg) was administered ip, alone or in combination with 0.5 g/kg ethanol. The take shock (T.S.—failure to respond to warning light or shock stimulus) responses and tranquilizer index (T.I.—value of the difference between performance and anxiety scores) were calculated. When ethanol was administered with 10 mg benzquinamide, a greater T.I. was effected, because the period of drug activity was extended. After 4 hr, the animals had still not returned to control performance, yet, analysis showed zero ethanol levels after 2 hr. As the drug dosage increased, there was greater ethanol potentiation—at 20 mg/kg, T.S. was too severe for tranquilization, and, at 30 mg/kg, profound inability to respond to stimuli was elicited.

425. Forney, R. B., and Hughes, F. W.
COMPARATIVE EFFECT IN HUMAN SUBJECTS OF CHLORMEZANONE, CHLORMEZANONE WITH ASPIRIN AND PLACEBO ON PERFORMANCE UNDER

DELAYED AUDITORY FEEDBACK (DAF).

Curr. Ther. Res. (New York), 6(10): 638-645 (7 ref.),

1964.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo
 – blood lev. – other drug lev. – psychol. perform. – CNS – analg., antipyret. – stimulants – *CAAAL-0
 A-0338.

The effects of chlormezanone (trancopal—100 mg, total 600 mg/day, or 200 mg, total 1200 mg/day), chlormezanone plus aspirin (trancogesic) (600 mg chlormezanone/day and 1800 mg aspirin/day), and placebo on performance were tested in 16 normal human volunteers, alone or in combination with ethanol (45 ml/68 kg). The symptom profile of the drugs suggested a subjective depression comparable to the sedative effect of ethanol, although chlormezanone and chlormezanone plus aspirin did not decrease performance parameters as did alcohol. After alcohol-drug combinations, there were more impairment symptoms checked in all cases than when the drugs were administered alone. The symptom severity scores after alcohol-drug combinations revealed that the lower dosage of chlormezanone or chlormezanone plus aspirin did not potentiate the symptomatology of alcohol; in fact, a reduction in severity was noted. Only after the 1200 mg/day chlormezanone dose was a moderate increase in symptom severity noted.

426. Forney, R. B., and Hughes, F. W.

EFFECTS OF ALCOHOL IN COMBINATION WITH DRUGS.

Traffic Digest and Review (Evanston), 12(5): 22-24 (14 ref.),

1964.

E – general – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mot. vehic. – humans – mammals – acute admin. – in vivo – mot. perform. – psychol. perform. – CNS
 – analg., antipyret. – barbiturates – tranquilizers – *CAAAL-0
 A-0699.

Recent experimental studies on man and animals, concerning the effects of alcohol-drug combinations, are reviewed. A comparative tranquilizer index of psychotherapeutic drugs (calculated from the difference between reduction in anxiety and decrease in performance), alone or in combination with ethanol, is presented. It is pointed out that the criteria for predicting the unexpected effects which may result from the drinking of ethanol are ambiguous, and the basis for anticipating drug side-effects are equally obscure. Studies of the influence on behaviour exercised by the combined use of ethanol and certain classes of drugs have really just begun.

427. Forney, R. B., and Hughes, F. W.

MEPROBAMATE, ETHANOL OR MEPROBAMATE-ETHANOL COMBINATIONS ON PERFORMANCE OF HUMAN SUBJECTS UNDER DELAYED AUDIOFEEDBACK (DAF).

J. Psychol. (Provincetown), 57: 431-436 (7 ref.),

1964.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev.
 – other drug lev. – psychol. perform. – CNS – tranquilizers – *CAAAL-0
 A-0323.

Verbal and arithmetical performance under an anxiety stimulus of delayed auditory feedback were measured in 8 subjects who received meprobamate (400 mg, 4 times/day), alcohol (100 proof whiskey, 45 ml/150 lb), or meprobamate-alcohol combinations. Alcohol in concentrations of 50 mg% in the blood effected a pronounced deficiency in performance. Meprobamate did not significantly affect performance, although there was a tendency toward impaired performance. Meprobamate plus alcohol produced greater impairment in several of the testing procedures than did either of the drugs alone.

428. Forney, R. B., and Hughes, F. W.

EFFECT OF CAFFEINE AND ALCOHOL ON PERFORMANCE UNDER STRESS OF AUDIOFEEDBACK.

Quart. J. Stud. Alcohol (New Haven), 26(2): 206-212 (15 ref.), 1965.
 E – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – other drug lev.
 – psychol. perform. – species or sex diff. – stimulants – *CAAAL-11185-J1 B-0167.

The effects of caffeine, alcohol, or both in combination on performance of 8 human subjects under stress of audiofeedback were evaluated in a double-blind study. Each subject was observed under four conditions: caffeine (500 mg caffeine citrate) plus placebo beverage, alcoholic beverage (100 proof whiskey, 45 ml/150 lb) plus placebo caffeine, alcoholic beverage plus caffeine, and placebo beverage plus placebo caffeine, and was graded on performance in 9 tests. In all tests, there was a decrease in performance when the blood alcohol concentration reached approximately 50 mg/100 ml. The reduction in performance by alcohol was significant in verbal output, reverse reading, and progressive counting. No complete picture of antagonism of ethanol-induced impairment of performance by caffeine was demonstrated.

429. Forney, R. B., and Hughes, F. W.
 ALCOHOL + DRUGS.
 Traffic Safety (Chicago), 67: 23-24, and 34-36 (35 ref.), 1967.
 E – general – presentation – DC (add., infra-add., unspec. incr.) – humans – mammals – blood lev.
 – mot. perform. – psychol. perform. – CNS – amphetamines – analg., antipyret. – hormones, hormone
 antag. – stimulants – tranquilizers – *CAAAL-0 B-0293.

This article is a shortened version of that appearing in Selzer, Melvin L., Gikas, Paul W., and Huelke, Donald F., eds., *The Prevention of Highway Injury* (Ann Arbor, 1967), pp. 70-77. The authors review several studies which have investigated the incidence and importance of the use of mood-modifying drugs, with and without alcohol, by the driving population, in an attempt to assess the role of these drugs in traffic mishaps. The authors also review some of their own work, which investigated the effects upon mental and motor performance of some of these drugs, alone and in conjunction with ethanol. They stress the importance of extending the routine blood analyses of accident victims, etc., to include analysis for a variety of drugs that can impair driving performance.

430. Forney, R. B.
 THE COMBINED EFFECT OF ETHANOL AND OTHER DRUGS.
 In: Selzer, Melvin L., et al., eds. *The Prevention of Highway Injury*. The proceedings of a symposium held April 19-21, 1967 in honour of the University of Michigan's Sesquicentennial Celebration, and sponsored by the University's Medical School and Highway Safety Research Institute. Ann Arbor, Michigan: Highway Safety Research Institute, 70-77 (34 ref.), 1967.
 E – general – presentation – DC (add., infra-add., unspec. incr.) – humans – mammals – blood lev.
 – mot. perform. – psychol. perform. – CNS – amphetamines – analg., antipyret. – hormones, hormone
 antag. – stimulants – tranquilizers – *CAAAL-0 B-0294.

The authors review several studies which have investigated the incidence and importance of the use of mood-modifying drugs, with and without alcohol, by the driving population, in an attempt to assess the role of these drugs in traffic mishaps. The authors also review some of their own work, which investigated the effects upon mental and motor performance of some of these drugs, alone and in conjunction with ethanol. They stress the importance of extending the routine blood analyses of accident victims, etc., to include analysis for a variety of drugs that can impair driving performance.

431. Forney, R. B., and Hughes, F. W.
 COMBINED EFFECTS OF ALCOHOL AND OTHER DRUGS.
 Springfield, Illinois: Charles C. Thomas, 124 pp. (318 ref.), 1968.
 E – review – DC (antidotal) – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec.
 incr.) – DC (sensit.) – med.-leg. – mot. vehic. – humans – mammals – other org. – blood lev. – other

drug lev. – mot. perform. – psychol. perform. – absorp., distrib., stor. – acid-base, blood pH, elect. – blood comp., sites, lymph – cardiovasc. – CNS – G.I. tract – glands – liver, kidney – metab. proc. – nerv. syst. – respir. – senses – skel., muscle, skin – alcohols – autocoids – autonomic agents – coagulants – gastrointest. agents – stimulants – unclass. ther. agents – *CAAAL-0 B-0295.

The authors explore a wide range of combinations of ethanol with various classes of drugs in common use. They discuss terminology pertinent to interaction, and the general pharmacological effects of ethanol. Ethanol interaction is discussed with reference to the following drugs: depressants (barbiturates, chloral hydrate, paraldehyde, glutethimide, bromides, ethinamate, methypylone, tranquilizers, and narcotics), stimulants (caffeine, amphetamines, monoamine oxidase inhibitors, methylphenidate, imipramine, amitriptyline, nortriptyline, and dexphenmetrazine), methanol, anticoagulants, hypoglycemic agents, antihistamines, disulfiram, ethacrynic acid, asparagine, dimethylsulfoxide, ephedrine, norepinephrine, acetylstrophanthidin, gamma-aminobutyric acid, *Coprinus atramentarius*, *Aesculus hippocastaneum*, glutamic acid, and glutamine. The authors conclude with a comment on legislation concerning alcohol-drug combinations.

432. Forney, R. B., and Hughes, F. W.

INTERACTION BETWEEN ALCOHOL AND PSYCHOPHARMACOLOGICAL DRUGS.

In: Trémolières, J., ed. *Alcohols and Derivatives. II*. International Encyclopedia of Pharmacology and Therapeutics, Section 20. Oxford: Pergamon Press, pp. 445-461 (54 ref.), 1970.

E – general – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – psychot. humans – mammals – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – species or sex diff. – CNS – liver, kidney – metab. proc. – nerv. syst. – antidepressants – barbiturates – enzymes – stimulants – tranquilizers – *CAAAL-0 B-0296.

The literature on the interaction between alcohol and tranquilizers, anti-depressants, and neuro-psychostimulants is reviewed. The terms "alcohol", "synergy", "synergism", "drug interaction", "tranquilizer", "anti-depressant", and "neuropsychostimulant" are defined. The authors state that the indistinct conclusions that must be made from the studies involving synergy of alcohol and psychopharmacological drugs result from a non-mechanistic approach. The pharmacology of single drugs is being explored in animals, including man, as well as at the cellular, enzymatic, and molecular level. The pharmacodynamic approach to drug-interaction studies involving alcohol is discouragingly insufficient.

433. Forsander, O. A.

INFLUENCE OF ETHANOL AND BUTYRALDOXIME ON LIVER METABOLISM.

Biochem. Pharmacol. (New York), 19: 2131-2136 (11 ref.), 1970.

E – exp. cont. – DC (sensit.) – mammals – acute admin. – in vivo – in vitro – blood comp., sites, lymph – liver, kidney – metab. proc. – *CAAAL-0 B-0933.

Experiments were conducted on intact rats and on rat liver slices. Albino rats (250-250 g) were given 1.2 g alcohol/kg ip as a 10% (v/v) saline sol, with or without butyraldoxime as 0.1% (w/v) of the sol. The ethanol effected a rise in acetaldehyde from 12.8-60.3 nmoles/g liver. Butyraldoxime had no effect on the acetaldehyde or blood glucose levels. Pretreatment with disulfiram, however, drastically increased acetaldehyde. In rat liver slices, ethanol oxidation was strongly inhibited by butyraldoxime, even at a concentration of 10^{-7} M. Ethanol, with or without butyraldoxime, had no effect on oxygen uptake. The oxime was competitive with ethanol, but not with NAD, and counteracted the effect of ethanol on the redox level of liver cytosol. At an oxime concentration of 10^{-5} M, ethanol had no effect on the lactate/pyruvate ratio; at this concentration, the ethanol oxidation rate was inhibited by 71%. It is concluded that, in the rat, butyraldoxime-induced intolerance to alcohol is not produced by increased acetaldehyde or any disturbances in liver metabolism.

434. Förster, A.
 DIE BLUTALKOHOLBEFUNDE UND IHRE VERWERTBARKEIT. [Blood alcohol determination and its value].
 Med. Klin. (Munich), 34(19): 633-635 (0 ref.), 1938.
 G – SEC – exp. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – mot. perform.
 – CNS – liver, kidney – respir. – stimulants – *CAAAL-860-A1 A-0700.

The author reports the results of blood alcohol determinations (Widmark method) in humans (no drug interaction involved). In addition, rabbits were put under alcohol anesthesia, and coramine (0.2 cc/kg) was injected at the peak of the alcohol effect. Immediately after the coramine, the animals became active; they attempted to run, and, after a few attempts, were able to do so. This state lasted for 15-20 min, and, after 45 min, the same condition as before existed, except that the animals reacted to stimuli. Respiration was more rapid after coramine. In the decreasing stage of intoxication, coramine had a permanent wakening effect—even 1 rabbit in severe shock with apnea could be awakened. The author concludes that coramine may decrease blood alcohol to a slight degree by increasing respiration, but, although the blood alcohol level is hardly influenced, the effect on the degree of intoxication is marked. Reference is also made to insulin, caffeine, camphor, hexetone, cardiazole, and theophylline.

435. Forster, F. C.
 CASE OF ACUTE ALCOHOLIC POISONING IN A CHILD AGED 4 YEARS:
 TREATMENT BY SALINE INJECTION: RECOVERY.
 Brit. Med. J. (London), 1: 1142-1143 (0 ref.), 1903.
 E – general – case hist. – DC (unchanged) – humans – CNS – liver, kidney – cardiovasc. agents –
 respir. agents – *CAAAL-0 A-1250.

The author describes the case of a 4 yr-old boy who had swallowed 2 oz of neat whiskey on an empty stomach. 45 min later, the boy was observed to be in a state of profound shock, characterized by: insensibility; low temperature; cold, clammy skin; shallow, sighing respirations; and an irregular, uncountable pulse. There was no vomiting, the corneal reflex was absent, and the pupils were dilated and reacted slightly to light. Initial treatment consisted of a hypodermic injection of strychnine sulphate (1/30 grain), siphoning of the stomach, and heat applications, followed by another injection of strychnine (1/50 grain) and digitalin (1/50 grain). Improvement, if any, was slight. Warm saline was then administered by rectum, in an attempt to dilute the poison and aid the kidneys in its elimination. Recovery was rapid—the boy drank hot tea after about 1/2 hr, and had resumed normal activity by the next day. Although the author does not attribute the patient's recovery entirely to the saline injection, since other remedies were adopted, he is convinced of its "great efficacy".

436. Fort, J.
 RECOMMENDED FUTURE INTERNATIONAL ACTION AGAINST ABUSES OF
 ALCOHOL AND OTHER DRUGS.
 Brit. J. Addict. (London), 62: 129-146 (0 ref.), 1967.
 E – SEC – general – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – hallucinogens – sed.,
 hypnot. – stimulants – *CAAAL-12548-L3 B-0297.

The state of the abuse of alcohol and other drugs throughout the world is discussed in detail. Countries which have a national problem of specific drug abuse are listed. The availability of drugs, legal or illegal, and the dangers of combined use of many of them, with or without alcohol, are discussed. A detailed program is presented for research and data collection on the abuse of alcohol and other drugs.

437. Foxell, A. W. H.
THREE CASES OF CARBON TETRACHLORIDE POISONING WITH ONE FATALITY.
 Brit. Med. J. (London), 1: 397 (4 ref.), 1951.
 E – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – humans – blood comp., sites, lymph – cardiovasc. – G.I. tract – liver, kidney – anti-infectants – *CAAAL-0 A-0701.

Of the 3 cases presented, one concerns a 30 yr-old man who accidentally consumed 14 ml carbon tetrachloride in lime juice, believing it to be gin. He later attended a party and drank gin and beer. The next morning, symptoms of poisoning began, and, 2 days after ingestion of the drug, he was admitted to hospital. Death from hepatic failure occurred 104 hr from the time of ingestion. The author considers that the alcohol, consumed quickly, increased the absorption of the carbon tetrachloride in the intestines. Successful recovery was made in the other 2 cases in which no alcohol was present.

438. Frahm, M., Löbkens, K., and Soehring, K.
DER EINFLUSS SUBCHRONISCHER ALKOHOLGABEN AUF DIE BARBITURAT-NARKOSE VON MEERSCHWEINCHEN. [The influence of subchronic administration of alcohol on barbiturate narcosis in guinea pigs].
 Arzneimittelforschung (Aulendorf), 12: 1055-1056 (1 ref.), 1962.
 G – ES – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – chronic admin. – in vivo – dose resp. – CNS – anesthetics – barbiturates – *CAAAL-10446-B2 A-0702.

Ethanol sol of 5, 10 or 15% concentration were applied to guinea pigs as the only source of liquid for 14 days. The onset and duration of narcosis with pentobarbital (20 mg/kg), methitural (70 mg/kg), and hexobarbital (50 mg/kg), were measured at intervals of 7 days. The onset of narcosis was slightly prolonged in proportion to the increased alcohol concentration, and the duration of narcosis was considerably reduced in proportion with the concentration of alcohol applied in all 3 series of experiments. The reduction of the narcotic effect of barbiturates was dependent on the continuous application of alcohol.

439. Francis, C. R.
THE USELESSNESS OF ALCOHOL IN SNAKE BITE.
 Medical Pioneer (London), 4(6): 80 (0 ref.), 1896.
 E – general – DC (unchanged) – humans – blood comp., sites, lymph – CNS – gastrointest. agents – *CAAAL-0 A-0703.

The author (writing for a temperance journal) expresses his views on alcohol as an antidote to snake venom. He states that snake venom, like alcohol, destroys the oxygen of the blood, and, therefore, science can have no faith in alcohol as an antagonist to venom. However, in some instances in which convulsions are the predominant symptom, he concedes that alcohol, especially when combined with opium, may be of possible benefit for its sedative effect. In conclusion, the author expresses the view that alcohol is a remedy which is discredited by science and insufficiently supported by experience.

440. Franco, S.
NEPHRITIC SYNDROMES CAUSED BY INDUSTRIAL POISONING WITH CARBON TETRACHLORIDE.
 New York J. Med. (New York), 36: 1847-1853 (24 ref.), 1936.
 E – SEC – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – blood comp., sites, lymph – cardiovasc. – G.I. tract – liver, kidney – respir. – anti-infectants – *CAAAL-0 A-0704.

This report reviews studies dealing with carbon tetrachloride poisoning, with special emphasis on renal damage. Two case histories are presented, one of which involved a heavy alcohol user. In the

latter case, the patient was severely intoxicated by exposure to carbon tetrachloride, while non-drinking fellow employees were apparently unaffected.

441. Frankenhaeuser, M., Fröberg, J., Goldberg, L., and Myrsten, A. -L.
EFFECTS OF ALCOHOL AS MODIFIED BY TRANQUILIZING DRUGS.
 University of Stockholm, Psychological Laboratories, Report No. 199, 9 pp. (17 ref.) November, 1965.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – CNS – nerv. syst. – tranquilizers – *CAAAL-0 B-0497.

0.55 g alcohol/kg body weight, in combination with a placebo, 800 mg meprobamate/70 kg, or 20 mg chlordiazepoxide/70 kg, were given to 8 male university students in a double-blind study, to determine if the tranquilizers modified alcohol effects or the time pattern of reactions. Blood alcohol concentrations, performance in objective tests (speed reaction, spokes test, and arithmetic), and subjective reactions were examined. There were no significant differences in blood alcohol concentrations between the 3 drug groups. Maximum values ranged between 0.64 and 0.66 mg/ml, and occurred about 30 min following alcohol intake. Performance was slightly impaired in all variables in the first post-alcohol trial, when alcohol and meprobamate were combined. Impairment was less for the alcohol-placebo and alcohol-chlordiazepoxide groups. Each mean curve for subjective intoxication estimates was highest at the first post-alcohol trial (75 min), and approached zero after 4 hr; this steeper decline, as compared to blood-alcohol curves, indicates a relative feeling of sobriety. Subjective evaluations were highest after alcohol-meprobamate, and lowest after alcohol-chlordiazepoxide, suggesting a possible increase and decrease, respectively, of subjective intoxication. The estimates of other mood variables in the 3 alcohol conditions showed small systematic deviations from the placebo condition.

442. Fraser, D. B.
 A CASE SHOWING THE ANTIDOTAL EFFECT OF ALCOHOL IN CARBOLIC-ACID POISONING.
 Medical Record (New York), 48: 741 (0 ref.), 1895.
 E – general – case hist. – DC (antidotal) – humans – skel., muscle, skin – anesthetics – *CAAAL-0 A-0705.

Case material is given concerning a woman who attempted suicide by swallowing 1 oz carbolic acid mixed with whiskey. The pulse was strong, regular, and not much accelerated; respiration was undisturbed; and the temperature slightly under 98° F. The patient regained consciousness in 8 hr, and was quickly able to answer questions. She declared that she had felt little pain or burning sensation, had slept soundly, and had had pleasant dreams. In a few days she was fully recovered. The author declares that alcohol is not only a pharmacological antidote, but a complete antidote, as its stimulatory effect counteracts the depression of carbolic acid poisoning.

443. Fraser, H. F., Wikler, A., Isbell, H., and Johnson, N. K.
 PARTIAL EQUIVALENCE OF CHRONIC ALCOHOL AND BARBITURATE INTOXICATIONS.
 Quart. J. Stud. Alcohol (New Haven), 18: 541-551 (8 ref.), 1957.
 E – exp. cont. – cross-tol. – drug-dep. humans – chronic admin. – in vivo – blood lev. – CNS – barbiturates – *CAAAL-8418-D1 A-0706.

In 10 morphine addicts in good physical health, opiates were withdrawn during two weeks, and secobarbital and pentobarbital, given orally 6 times daily (1.06 to 2.3 g daily) for 22 to 44 days, were substituted. Alcohol was then given in place of the barbiturates (between 312 and 460 ml of alcohol

daily), and withdrawn after 14 days. The effects are described in detail, and it is concluded that, while alcohol does not meet all the rigorous requirements of a complete substitute for barbiturates, it is certainly a potent partial substitute. A gradual reduction of sedatives administered to alcoholics as substitution therapy should make possible a gradual, rather than a precipitous, loss of physical dependence on alcohol, thus preventing the appearance of convulsions and delirium.

444. Freeman, J., and Schulman, M. P.

REACTIONS OF CHLORAL HYDRATE AND ETHANOL WITH ALCOHOL DEHYDROGENASE FROM HUMAN LIVER.

Fed. Proc. (Bethesda), 29(2): 275Abs. (0 ref.),

1970.

E – abst. – exp. cont. – DC (add., infra-add. unspec. incr.) – acute admin. – in vivo – liver, kidney – metab. proc. – sed., hypnot. – *CAAAL-0 B-0934.

The basis of the chloral hydrate (CH)-ethanol synergism has been thought to be inhibition of alcohol dehydrogenase (ADH) by CH and its reduced metabolite, trichloroethanol (TCE). Present results permit a formulation which incorporates the fact that TCE has greater hypnotic potency than CH. ADH was purified from human liver by column chromatography, and measured as NADH formation from ethanol and NAD at pH 9.3 (condition I); the purified enzyme formed 1 μ M NADH/min/mg protein. CH reduction to TCE by ADH in the absence of ethanol was followed by disappearance of added NADH (II). The coupled redox between ethanol and CH was studied by measuring acetaldehyde (AcH) evolution from the reaction mixture containing the substrates and limited NAD in potassium phosphate pH 7.3 (III). The oxidation of ethanol to AcH was 23 times greater in the presence of CH than in its absence under condition III, and 10 times greater than under condition I. The calculated reduction of CH in III in the presence of ethanol was 7 times greater than that observed with NADH without ethanol as in II. The acceleration of CH reduction to its pharmacologically active form, TCE, by a coupled redox of CH and ethanol catalyzed by ADH, may provide the molecular basis for the synergism between the 2 drugs.

445. Frey, H. -H., Doenicke, A., and Jäger, G.

VERLAUF DER SERUMSKONZENTRATION WÄHREND DER ERSTEN 24 STD. NACH INTRAVENÖSEN KURZNARKOSEN MIT EINEM THIOBARBITURAT. [The course of serum concentration during the first 24 hours after intravenous narcosis with a thiobarbiturate].

In: *Mitteilungen der Deutschen Gesellschaft für Verkehrsmedizin. X. Referat und Diskussion anlässlich der Arbeitstagung der Sektion "Arzneimittel und Verkehr".* [Communications of the German Society for Traffic Medicine. X. Reports and discussion arising out of the working session of the Section "Drugs and Driving"]. Bad Oeynhausen, West Germany, March 25-26, 1961. Mainz: Geschäftsstelle der Deutschen Gesellschaft für Verkehrsmedizin e.V., pp. 17-18 (0 ref.), 1961.

G – SEC – presentation – cross-tol. – humans – CNS – barbiturates – *CAAAL-0 A-0707.

2 groups of subjects (clinical patients and volunteers) were used to determine the influence of post-narcosis serum concentrations on the driving ability of ambulatory patients. The concentrations in both groups were determined photometrically 2, 4, 8, and 24 hr after iv injections of 1-1.5 g thiobutabarbitol sodium, and, secondly, after 0.2 g butabarbitol po. The mean concentration of thiobutabarbitol, determined 24 hr after injection, was 5 μ g/ml, and the plasma concentration of butabarbitol 2-6 hr after injection was 4 μ g/ml. The authors feel that cases of an "acute tolerance" to the hypnotic effect of barbiturates may be broken by low doses of drugs or alcohol, and they endorse Soehring's recommendation of a 24-hr driving abstention following iv short narcosis.

446. Fridman, V.

UDRUŽENO DEJSTVO ALKOHOLA I DRUGIH ALKOHOLNIH SUPSTANCIJA

(OLOVA, ŽIVE I DR.). [Combined effect of alcohol and other toxic substances (lead, mercury, etc.)].

Alkoholizam (Belgrade), 8(1): 51-55 (16 ref.), 1968.
 Se – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – mammals – absorp., distrib., stor. – blood comp., sites, lymph – cardiovasc. – CNS – metab. proc. – respir. – miscellaneous – unclass. ther. agents – *CAAAL-0 B-0298.

The author reviews data in the literature on the behaviour of alcohol in the animal organism subjected to toxic substances. In clinical and experimental studies, alcohol considerably reduced tolerance to lead poisoning and aggravated its toxic symptoms, especially in relation to functions of the CNS and the so-called lead colic phenomena. In other tests, ethanol potentiated the toxic effects of nitrogen compounds. On the other hand, ethanol reduced absorption of mercury vapours from the lungs (75 to 57%). Details on the reaction mechanism are given. Also mentioned is the interaction of *Coprinus atramentarius* with alcohol, and the resultant antabuse-like reaction. The interaction mechanism leading to the antabuse syndrome is explained.

447. Friedländer, A.
 ZUR KLINIK DER INTOXICATIONEN MIT BENZOL- UND TOLUOL-DERIVATEN, MIT BESONDERER BERÜCKSICHTIGUNG DES SOG. ANILINISMUS (VORLÄUFIGE MITTEILUNG). [Clinical aspects of intoxications with benzol and toluol derivatives, with special consideration of the so-called anilism (preliminary communication)].
 Neurologisches Zentralblatt (Leipzig), 19: 155-162, and 294-297 (1 ref.), 1900.
 G – general – DC (add., infra-add., unspec. incr.) – humans – CNS – unclass. ther. agents – *CAAAL-0 A-0708.

A general discussion is given concerning the use of aniline in industrial plants in various parts of the world. The author refers to literature on poisoning of workers by benzene derivatives. Based on data of various investigators and his own observations, the following guidelines are given: 1. Benzene and toluene are poisonous substances affecting the CNS, as well as the circulatory system. 2. Prior alcohol intake potentiates the action of these derivatives. 3. Latent intoxications are brought out by alcohol. 4. Alcohol is extremely dangerous to workers exposed to the substances in question.

448. Friedman, S. L., and Ingalls, J. W., Jr.
 A NOTE ON THE TILTING-PLANE TECHNIQUE FOR MEASURING THE PERFORMANCE OF RATS IN RELATION TO THE DEGREE OF THEIR ALCOHOL INTOXICATION.
 Quart. J. Stud. Alcohol (New Haven), 21(2): 217-222 (2 ref.), 1960.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – mot. perform. – tranquilizers – *CAAAL-8764-J2 A-0709.

The reliability of the tilting-plane method for quantitating the effect of tranquilizers and alcohol on gross reactions and motor coordination was investigated in 72 male rats. The technique proved capable of distinguishing degrees of alcohol intoxication (1875 mg to 7500 mg/kg), and of demonstrating synergism between alcohol (1875 mg/kg) and chlorpromazine (20 mg/kg). The addition of 20 mg/kg chlorpromazine to the alcohol had the same effect as would increasing the dose of alcohol by 675 mg/kg, i.e., a 36% increase in sliding angle.

449. Fröberg, J.
 EFFEKTER AV ALKOHOL OCH TVÅ CENTRALNERVÖST VERKANDE DROGER PÅ REAKTIONSTID. [Effects of alcohol and two drugs acting on the central nervous system on reaction time].
 Dissertation, Institute of Psychology of the University of Stockholm, Sweden, 7 pp. + 11 pp. appendix (6 ref.), 1963.
 S – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – CNS – tranquilizers – *CAAAL-0 A-0710.

Reaction times were studied in 8 male students after: a) placebo, b) placebo plus alcohol (0.55 ml/kg absolute alcohol), c) librium (20 mg/70 kg—0.3 mg/kg), or d) meprobamate (800 mg/70 kg—11.5 mg/kg) plus alcohol. Blood samples were taken 30, 60, 90, 130, 180, 240, 300, 360, and 420 min after alcohol ingestion. Widmark's method was used. No significant effect of alcohol plus drug, in comparison with alcohol plus placebo was established. The mean and maximum values of blood alcohol concentration (in mg/ml) and registered reaction times (in min) of the combinations are plotted and tabulated.

450. Frommel, E., Ledebur, I. von, Joye, E., Duda, M., and Seydoux, J.
THE ANTIDOTAL ACTION OF NALORPHINE IN COMBATTING ALCOHOL INDUCED BRADYPNEA IN THE RABBIT.
 Med. Exp. (Basel), 9: 38-40 (7 ref.), 1963.
 E – FS – GS – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – respir. – hallucinogens – *CAAAL-10934-D2 A-0711.

Nalorphine (10 mg/kg) significantly antagonized ethanol (1 cc/kg)-induced bradypnea in the rabbit. This phenomenon was not due to the effects of an iv injection per se, because controls were injected with an equal quantity of physiological sol. The authors consider that the fact that nalorphine administered alone induces tachypnea, while at the same time counteracting the effects of morphine- and alcohol-induced bradypnea, constitutes a problem which bears upon the complex pharmacodynamics of the analeptic substances. Nevertheless, the fact remains that this antidotal action goes beyond the framework of the hypothesis of alcaloid competition, and may be classed in the same group of effects as the action of nalorphine in relation to chlorpromazine, meprobamate, and librium.

451. Frommel, E., Seydoux, J., and Fasel, M.
LA MORPHINE ET LE PHÉNOBARBITAL MODIFIENT-ILS LE TAUX DE L'ALCOOLÉMIÉ PROVOQUÉE CHEZ LE COBAYE? [Do morphine and phenobarbital modify induced blood alcohol levels in the guinea pig?].
 Experientia (Basel), 19(11): 602-603 (8 ref.), 1963.
 F – ES – exp. cont. – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – CNS – metab. proc. – analg., antipyret. – barbiturates – gastrointest. agents – *CAAAL-10971-D2 A-0712.

5 cc/kg 60% ethanol was administered to guinea pigs, simultaneously with 13.5 mg/kg morphine sc or 30 mg/kg phenobarbital po. Blood alcohol levels were determined spectrophotometrically 30, 60, and 90 min later; the maximum level was reached after 90 min. Neither the morphine nor the phenobarbital significantly influenced the blood alcohol level. The results confirm those of Dille, James M., and Ahlquist, Raymond P. (J. Pharmacol. Exp. Ther. (Baltimore), 61(4): 385-392, 1937), and contradict those of Whittlesey, Philip (Johns Hopkins Hospital, Bulletin (Baltimore), 95: 81-89, 1954). In the guinea pig and the rat, it appears that the inhibition of oxidative phenomena by opium alkaloids and barbiturates exerts no significant influence on blood alcohol levels.

452. Frommel, E., Seydoux, J., and Ledebur, I. von
QUEL EST L'ANTIDOTE DE L'EXCITATION PSYCHOMOTRICE DE L'ÉTHYLIQUE? PHÉNOBARBITAL MORPHINE, LIBRIUM OU CHLORPROMAZINE? ETUDE EXPÉRIMENTALE. [Which is the antidote for the psychomotor excitation of the alcoholic? Phenobarbital, morphine, librium or chlorpromazine? Experimental study].
 C.R. Soc. Biol. (Paris), 157(3): 526-529 (3 ref.), 1963.
 F – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – blood lev. – CNS – analg., antipyret. – barbiturates – tranquilizers – *CAAAL-10971-D2 A-0286.

The effects of 30 mg/kg phenobarbital po, 13.5 mg/kg morphine sc, 10 mg/kg librium po, and 30 mg/kg chlorpromazine sc, in combination with 5 cc/kg 60% ethanol po, were determined in guinea pigs. Phenobarbital and morphine considerably increased ethanol sleeping time, but librium had no significant effect. Chlorpromazine had no effect in guinea pigs weighing less than 800 g, but significantly shortened sleep when weight was more than 800 g. Librium and chlorpromazine thus would seem to be better antidotes of psychomotor excitation due to alcohol than the other 2 drugs.

453. Frommel, E., and Seydoux, J.

DE L'EFFET DE L'ÉTHANOL SUR L'ENCÉPHALE: BILAN DES TESTS DITS DE NEUROPHARMACOLOGIE CHEZ L'ANIMAL. [On the effect of ethanol on the brain: results of so-called neuropharmacological tests in animals].

Helv. Physiol. Pharmacol. Acta (Basel), 22: 34-38 (9 ref.),

1964.

F – ES – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – mot. perform. – psychol. perform. – CNS – analg., antipyret. – anesthetics – barbiturates – tranquilizers – *CAAAL-11072-D2 A-0713.

Experiments were conducted on guinea pigs and mice. The effects of simultaneous administration of 5 ml/kg 60% ethanol po and phenobarbital (30 mg/kg po), librium (10 mg/kg po), chlorpromazine (30 mg/kg sc), morphine (13.5 mg/kg sc), ether (7 ml), or hexobarbital (75 mg/kg ip) on sleeping time; of 5 ml/kg ethanol on hypothermia caused by 0.6 ml/kg pyrexal ip, 20 mg/kg morphine sc, 30 mg/kg chlorpromazine sc, or 10 mg/kg librium po; of ethanol and 5 mg/kg morphine sc on perception of pain; of 5 ml/kg ethanol on motor excitation caused by 150 mg/kg nikethamide sc; of ethanol on electroshock in the guinea pig; of ethanol on the toxicity of strychnine and cardiazol (no dosages given); and of ethanol on psychomotor excitation caused by 10 mg/kg amphetamine sc, were determined. Acute intoxication by ethanol was shown to have a generalized depressing action on the CNS, e.g., it greatly reduced the agitation caused by nikethamide sc, the effects of electroshock were lessened in guinea pigs, and cortical and subcortical structures were affected by ethanol in psychomotor excitation due to amphetamine. A functional depression of such extent may partly explain the psychological behaviour of the drinker.

454. Fuchs, G.

DIE WIRKUNG VON PERVITIN IN THERAPEUTISCHER DOSIS BEI PATIENTEN, DIE ZUM AUFTRETEN EINES PATHOLOGISCHEN RAUSCHES NEIGEN. [The action of pervitin in therapeutic doses in patients inclined towards pathological drunkenness].

Dissertation, Medical Faculty of Humboldt University of East Berlin, East Germany, 78 pp. (39 ref.), 1963.

G – exp. cont. – DC (decrease) – drug-dep. humans – acute admin. – in vivo – mot. perform. – psychol. perform. – cardiovasc. – nerv. syst. – amphetamines – *CAAAL-0 A-0714.

8 persons inclined to pathological intoxication were tested for circulation and psychophysical performance under controlled conditions with pervitin (0.009 g) and alcohol (1.3-1.6°/oo). Appreciable differences were found in the circulation with alcohol-pervitin, as opposed to alcohol alone. Pervitin improved qualitative performance with time. Outer attentiveness improved markedly 270 min after pervitin, but inner attentiveness corresponded to the control test. Coordination and movements were undisturbed 150 min after alcohol-pervitin, as opposed to alcohol alone. The horizontal nystagmus test was only slightly influenced by pervitin; it was positive after 90 min. No change in reflex behaviour was noted.

455. Fühner, H.

UNTERSUCHUNGEN ÜBER DEN SYNERGISMUS VON GIFTEN. I. DIE KOMBINATION VON HERZGIFTEN (METHYLVIOLETT) MIT ALKOHOL UND GLYZERIN. [Investigations of the synergism of poisons. I. The combination of heart poisons

(methyl violet) with alcohol and glycerin].

Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 69: 29-44 (10 ref.), 1912.

G – exp. comp. – DC (add., infra-add., unspec. incr.) – other org. – acute admin. – in vivo – absorp., distrib., stor. – cardiovasc. – anti-infectants – *CAAAL-0 A-0715.

This study reports the first successful quantitative determination of the amount of methyl violet capable of arresting the frog heart. 2 mg methyl violet sc arrested the heart after 8 hr, and 4 mg after 4 hr. Glycerin or alcohol added to methyl violet accelerated the arrest, but in different ways—glycerin increased the rate of absorption, alcohol reduced it. That the alcohol-methyl violet combination nevertheless accelerated the heart arrest was due to the synergistic effect. In the glycerin-treated frogs, the amount of dyestuff in the heart was almost unchanged, compared with the water-treated frogs; in the alcohol-treated frogs, the amount was markedly reduced. The synergism between alcohol and methyl violet was confirmed in experiments on the isolated frog heart.

456. Fühner, H.

ALKOHOL-VERGIFTUNG, MEDIZINALE. [Alcohol poisoning, medical].

Sammlung von Vergiftungsfällen (Berlin), 1: 173-174 (2 ref.), 1930.

G – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – respir. – analg., antipyret. – sed., hypnot. – *CAAAL-0 A-0716.

In order to cure a cold, a woman ingested 0.25 g of quinal, 50 mg of noctal, and a bottle of wine. Acute alcohol poisoning with respiratory failure and threat of respiratory paralysis were the immediate effects. Recovery followed after constant stimulation of respiration and skin. This is the earliest reported case of alcohol-barbiturate interaction in medical literature, according to Eerola, Risto (Ann. Med. Exp. Biol. Fenn. (Helsinki), 39(Suppl. 3): 1-70, 1961).

457. Fuhrman, F. A.

THE EFFECT OF BODY TEMPERATURE ON DRUG ACTION.

Physiol. Rev. (Bethesda), 26: 247-274 (208 ref.), 1946.

E – SEC – review – DC (unchanged) – mammals – other org. – metab. proc. – hormones, hormone antag. – *CAAAL-4706-A2 A-0717.

The literature on the subject is reviewed and discussed. The rate of metabolism of alcohol in homeotherms is higher in small animals with high metabolic rates than in larger animals; however, in a given species, attempts to increase the rate of alcohol metabolism by increasing the metabolic rate with thyroxin or dinitrophenol, or by exposure to cold, did not significantly alter the oxidation rate. Elevation of body temperature by means of diathermy produced slight increases in the metabolic rate. The reported data, taken together with those demonstrating failure to increase the rate of combustion of alcohol by an increase in metabolic rate, show that in homeotherms, as well as in poikilotherms, the rate of alcohol metabolism is dependent upon body temperature.

458. Gabriel, C. L.

ANTAGONISM BETWEEN ALCOHOL AND STRYCHNINE.

Australasian Medical Gazette (Sydney), 11: 197-198 (1 ref.), 1892.

E – SEC – general – DC (decrease) – humans – stimulants – *CAAAL-0 A-0718.

The author draws attention to the antagonism of strychnine and alcohol, and states that the use of alcohol in cases of snake-bite poisoning treated with strychnine is thus contraindicated. The question is also posed whether the vomiting occasionally encountered in snake-poisoning is due to stimulation of the vomiting centre in the medulla, or to elimination of the poison by the mucous membrane of the stomach and irritation of the terminal fibres of the pneumogastric in that viscus.

459. Gaede, D., and Kiese, M.
 PHARMAKOLOGISCHE UNTERSUCHUNGEN ÜBER M-DINITROBENZOL. V.
 MITTEILUNG. M-DINITROBENZOL UND ALKOHOL. [Pharmacological studies on
 m-dinitrobenzene. V. m-Dinitrobenzene and alcohol].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 206:
 569-583 (10 ref.), 1949.
 G – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin.
 – chronic admin. – in vivo – species or sex diff. – blood comp., sites, lymph – CNS – nerv. syst. –
 miscellaneous – *CAAAL-5722-D2 A-0719.

16 dogs received 10 mg/kg of m-dinitrobenzene (m-d) in 2% sol in oil sc, and the physiological effects were tested before and 2 days after the injection. In a second experiment, dogs received 3 ml/kg of 98% alcohol in 50% sol by stomach tube. In a third experiment, dogs received daily 0.2-1.0 mg/kg of m-d for 15-227 days, then 3 ml/kg of alcohol. More experiments are described, some of them with rats. Conclusion: No synergism between m-d and alcohol was shown in dogs. In rats, however, synergism was shown both in the effect of alcohol subsequent to m-d, and in the effect of m-d subsequent to alcohol.

460. Gaisbauer, G.
 ZUR FRAGE DES MEDIKAMENT-ALKOHOLSYNERGISMUS IN DER
 VERKEHRSRECHTLICHEN PRAXIS. [The question of drug-alcohol synergism in the practice
 of traffic law].
 Neue Juristische Wochenschrift (Berlin), 33: 1504-1505 (15 ref.), 1967.
 G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – blood lev. – CNS –
 barbiturates – sed., hypnot. – stimulants – tranquilizers – *CAAAL-0 B-0299.

In connection with a court decision (Oberlandesgericht Hamm: Urteil vom 3.11.66; Deutsches Auto-recht (Munich), 36: 141-142, 1967, and Blutalkohol (Hamburg), 4(4): 221-223, 1967), the author discusses the combined action of alcohol and drugs. An enhanced alcohol effect is to be expected by simultaneous intake of alcohol and drugs of the following groups: tranquilizers, antihistamines, analgetics, opiates, hypnotics, sedatives, stimulants, and others. Use of sedatives after consumption of alcohol may result in an increased level of blood alcohol, unconsciousness, and even death. Intake of a small quantity of alcohol many hr after a dose of 200 mg butabarbital may result in a state of complete drunkenness.

461. Gallard, T.
 DE L'EMPOISONNEMENT PAR LA STRYCHNINE. [Strychnine poisoning].
 Annales d'Hygiène Publique et de Médecine Légale, (Paris), (Ser. 2) 23: 368-420, and (Ser. 2) 24:
 129-188 (75 ref.), 1865.
 F – SEC – general – case hist. – DC (antidotal) – humans – psychol. perform. – cardiovasc. –
 stimulants – *CAAAL-0 A-0720.

The medico-legal aspects of strychnine poisoning and its symptoms are discussed. Treatment of strychnine poisoning, including alcohol treatment, is listed. Case material is cited in which alcohol (30 g) and aqueous cinnamon bark brought about recovery from strychnine poisoning symptoms (weak pulse, white pallor, and reduced intellectual comprehension). This treatment was followed up with a bottle of Burgundy wine, whereupon the patient resumed his normal work and seemed fully recovered. It is conjectured that the efficacy of alcohol as an antagonist to strychnine depends upon the amount of strychnine absorbed in the body.

462. Galloway, D. H.
STRONG CARBOLIC ACID USED BY MISTAKE AS A THROAT SPRAY.
 Laryngoscope (St. Louis), 9: 114-115 (0 ref.), 1900.
 E – general – case hist. – DC (antidotal) – respir. – anesthetics – *CAAAL-0 A-0721.
- A case history of severe tonsillitis and pharyngitis treated with carbolic acid (1 oz via atomizer), which was mistaken for hydrogen peroxide, is given. Upon discovering the error, the physician filled the atomizer with equal parts of alcohol and water, and sprayed the throat and mouth of the patient 10-12 times within 15-20 min. During this time, the patient was choking and almost suffocated. When he was able to breath again, the treatment was stopped, and a mucilage of slippery elm bark was prescribed for drinking at frequent intervals. Complete recovery from the carbolic-acid-induced inflammation and the tonsillitis was effected in one week, the patient never suspecting the error.
463. Gamble, N. J.
THE EFFECT OF ALCOHOL AND AMPHETAMINE UPON BENDER-GESTALT REPRODUCTIONS.
 M.A. Thesis, Faculty of Graduate Studies of the University of Alberta at Edmonton, Canada, 103 pp. (37 ref.), 1964.
 E – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – mot. perform. – CNS – *CAAAL-0 A-0722.
- The effect of alcohol and amphetamine upon the Bender-Gestalt test was investigated in a group of pharmacy and medical students. One test group of 25 subjects was given the Bender-Gestalt over a 4-week period in the following sequence: pretest, 2 administrations of alcohol and amphetamine, and post-test. Each subject received both drug combinations in random order. A control group of 25 subjects received 4 administrations of the Bender-Gestalt for 4 weeks under normal conditions. Alcohol (1.2 gm/kg) diminished inhibition or cortical control, and, when amphetamine (15 mg) was used in combination with alcohol, the effect was significantly intensified.
464. Ganz, V.
THE ACUTE EFFECT OF ALCOHOL ON THE CIRCULATION AND ON THE OXYGEN METABOLISM OF THE HEART.
 Amer. Heart J. (St. Louis), 66(4): 494-497 (16 ref.), 1963.
 E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – cardiovasc. – CNS – anesthetics – *CAAAL-10854-D2 A-1395.
- Experiments were conducted on 10 dogs under thiopentone anesthesia, to obtain information on the possible mechanism of action of alcohol on anginal pain. 96% alcohol was administered iv by a constant-infusion pump in doses of 51-86 mg/kg for 20 min. Measurements were made of cardiac output, coronary sinus outflow, aortic pressure, pulse rate, and oxygen content of arterial and coronary sinus blood, at 10-min intervals, commencing 10 min before infusion. There was a decrease in stroke volume, and in external work of the left ventricle due to diminished cardiac output. Arterial blood pressure did not change, and there was a rise in peripheral resistance. Coronary sinus outflow increased slightly, as a result of a fall in coronary resistance. Perfusion pressure remained unchanged. Myocardial oxygen consumption increased, paralleling a rise in coronary flow. The oxygen content of the coronary sinus, and the amount of oxygen extracted by the myocardium, did not change. The findings show no evidence for a specific effect of alcohol on the defect present in angina pectoris.
465. Gardner, G. H., Grove, R. C., Gustafson, R. K., Maire, E. D., Thompson, M. J., Wells, H. S., and Lamson, P. D.
STUDIES ON THE PATHOLOGICAL HISTOLOGY OF EXPERIMENTAL CARBON TETRACHLORIDE POISONING.

Johns Hopkins Hospital, Bulletin (Baltimore), 36: 107-133 (18 ref.), 1925.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood comp., sites, lymph – G.I. tract – liver, kidney – respir. – anti-infectants – *CAAAL-0 A-0723.

The authors investigated the toxicity and pharmacology of carbon tetrachloride, as well as the pathological changes following the administration, in dogs and rabbits. Among the results was the finding that simultaneous oral administration of alcohol with 4 cc CCl₄/kg to adult dogs markedly increased the toxicity of CCl₄, as shown by the tremendous liver destruction and high death rate.

466. Gay, A., Montale, P., and Peris, G.
RICERCHE CLINICHE E SPERIMENTALI CON UN NUOVO CHEMIOTERAPICO: IL FURALTADONE. [Clinical and experimental investigation of a new chemotherapeutic agent: furaltadone].
Arch. Maragliano Pat. Clin. (Genoa), 16: 621-632 (12 ref.), 1960.
I – SEC – exp. – case hist. – DC (sensit.) – humans – other org. – chronic admin. – in vivo – cardiovasc. – CNS – metab. proc. – anti-infectants – *CAAAL-0 A-1450.

The therapeutic efficacy of an antibiotic, furaltadone, was investigated in tests on bacterial strains and on patients being treated for various microbial afflictions. 16 patients received 1 or 1.5 g furaltadone/day for a period of 4-20 days. Results of the treatment and eventual side-effects or other toxic manifestations were assessed. In 14 of the cases, treatment was successful. In 6 cases, side effects occurred with the 1.5 g furaltadone dose, manifested by nausea (rarely accompanied by vomiting) in 5 cases, and, in the other case, by reddening and a heat sensation in the face, together with slight dysphagia; in this sixth patient, the suspension of a modest quantity of alcohol (wine) in the diet brought about the immediate disappearance of symptoms. In 3 others, the disorders were alleviated by a reduction of the furaltadone dosage to 1 g/day. Further evidence of alcohol intolerance during furaltadone treatment is adduced from the literature, the phenomenon being attributed to the interference of furaltadone with the oxidation of alcohol in cerebral tissue.

467. Gebhart, G. F., and Mitchell, C. L.
THE EFFECTS OF ALCOHOL IN COMBINATION WITH THREE CENTRALLY ACTING AGENTS.
Pharmacologist (Washington), 10(2): 214 (0 ref.), 1968.
E – abst. – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – barbiturates – tranquilizers – *CAAAL-0 B-0300.

Chlordiazepoxide, chlorpromazine, phenobarbital, or ethyl alcohol (50% v/v) were administered ip to male albino mice, and PD₅₀'s determined for paralysis on an inclined plane. 1/2 of the PD₅₀ of ethanol was then given in combination with 1/2 the PD₅₀'s of the other three drugs. Only chlordiazepoxide demonstrated potentiation. Also, effects of the above-mentioned drugs in combination with ethanol were examined, utilizing loss of righting reflex as the criterion. Potentiated effects were seen for all three agents; however, the number of delayed deaths for ethanol-drug combinations was significantly less than for ethanol alone.

468. Gebhart, G. F., Plaa, G. L., and Mitchell, C. L.
THE EFFECTS OF ETHANOL ALONE AND IN COMBINATION WITH PHENOBARBITAL, CHLORPROMAZINE, OR CHLORDIAZEPOXIDE.
Toxic. Appl. Pharmacol. (New York), 15(2): 405-414 (21 ref.), 1969.
E – exp. cont. – exp. comp. – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – mot. perform. – absorp., distrib., stor. – CNS – liver, kidney – barbiturates – tranquilizers – *CAAAL-0 B-0516.

The interactions between ethanol (E) and phenobarbital sodium (Pb), chlorpromazine hydrochloride (CPZ) and chlordiazepoxide hydrochloride (CDP) were studied in mice. All drugs were administered ip in doses of 0.1 ml drug sol/10 g body wt. The PD_{50} was determined by the failure of the mice to remain on a 60° inclined screen. 1/2 of the PD_{50} of CPZ, CDP, or Pb was given in combination with 1/2 the PD_{50} of E, such that the times of peak effect of the combined drugs coincided. The ED_{50} for E, required to produce a median loss of righting reflex (LRR_{50}), was determined, and 1/2 the LRR_{50} dose of E was studied in combination with 1/2 the PD_{50} of CPZ, CDP, Pb, and E. The blood level of ethanol (BLE) was determined at 5, 15, 30, 60, and 120 min after E administration. Only CPZ and E produced a supra-additive effect in the inclined screen experiment, whereas Pb and CPZ demonstrated an addition of effects. All 3 drugs showed supra-addition with the much larger LRR_{50} dose of E. It is concluded that none of the 3 drugs produce their effects by increasing the BLE, since the BLE was not found to be elevated at the time of the behavioral effects.

469. Genest, K., Coldwell, B. B., and Hughes, D. W.
 POTENTIATION OF ETHANOL BY *COPRINUS ATRAMENTARIUS* IN MICE.
 J. Pharm. Pharmacol. (London), 20: 102-106 (13 ref.), 1968.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (sensit.) – mammals – acute
 admin. – in vivo – mot. perform. – CNS – miscellaneous – *CAAAL-13049 B-0517.

The authors investigated the question whether 2 species of mushrooms, *Coprinus atramentarius* and *Coprinus comatus*, increase the effect of alcohol in mice. Raw forms (4.5 g/kg) of these mushrooms were fed po to groups of 10 mice. Ethanol (5 g/kg) was given at different times before and after mushroom feeding. Control groups had only ethanol or 1 of the mushrooms. Toxic effects were greatest when ethanol was given 3-6 hr after mushroom feeding, less with simultaneous administration or after 16 hr, and practically absent after 24 hr or after reversing the feeding order; the *C. comatus*-ethanol group showed only minor differences from the ethanol-control group. The average sleeping duration in the *C. atramentarius*-ethanol group was almost double that of the ethanol control group, although induction times were quite similar. Intoxication symptoms (e.g., ataxia, loss of righting reflex, and sedation) were nearly identical for both cooked and raw mushrooms, when administered before alcohol.

470. Geraghty, F. J., and Rogers, W. A.
 CARBON TETRACHLORIDE POISONING WITH REPORT OF A CASE.
 W. Virginia Med. J. (Charleston), 43: 242-246 (10 ref.), 1947.
 E – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – absorp., distrib., stor. –
 blood comp., sites, lymph – cardiovasc. – CNS – G.I. tract. – liver, kidney – respir. – skel., muscle,
 skin – anti-infectants – *CAAAL-0 A-0724.

The dangers of exposure to carbon tetrachloride are discussed, and a case history of poisoning is presented. The consumption of alcohol in considerable amounts was found to increase the toxicity of this compound, regardless of whether it was inhaled or ingested, by increasing the rate and degree of absorption from the intestinal tract. The pathology was that of acute hepatitis and toxic nephrosis. The treatment was symptomatic, directed at protection of the liver and cardiovascular system and at combatting anuria and acidosis.

471. Gervais, D. M.
 NOTE SUR LES BONS EFFETS DE L'AMMONIAQUE DANS L'IVRESSE. [Note on the
 beneficial effects of ammonia on intoxication].
 Bulletin Général de Thérapeutique Médicale, Chirurgicale, Obstétricale et Pharmaceutique (Paris),
 18: 35-37 (0 ref.), 1840.
 F – general – case hist. – DC (antidotal) – humans – CNS – respir. – stimulants – *CAAAL-0
 A-0725.

The author refers to some investigators who question the validity of ammonium therapy in alcoholic intoxications, and asserts its usefulness by giving 4 case histories in which ammonia proved an effective agent for inebriety. The first case involved a man intoxicated by brandy, who was found in a deep coma without any signs of sensitivity. An open bottle of ammonia on a piece of cotton were applied to the man's lips and eyelids. The man stirred, and swallowed some sweetened water containing 15 drops of ammonia. His eyes opened and closed. He was given another dose of ammonia as above. 20 min later he was fully revived.

472. Gessner, P. K., and Cabana, B. E.
THE EFFECT OF ETHANOL ON CHLORAL HYDRATE HYPNOSIS IN MICE.
 Fed. Proc. (Bethesda), 23: 348 (0 ref.), 1964.
 E – abst. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo
 – dose resp. – sed., hypnot. – *CAAAL-0 A-0726.

Experiments demonstrated ethanol potentiation of chloral hydrate hypnosis in mice. The median effective doses for loss of righting reflex following ip administration were: chloral hydrate—248 mg/kg, ethanol—2750 mg/kg, chloral hydrate in presence of 60 mg/kg ethanol—203 mg/kg, and ethanol in presence of 218 mg/kg chloral hydrate—29 mg/kg. It is concluded that the potentiation is not due to simple addition of hypnotic effects.

473. Gessner, P. K., and Cabana, B. E.
THE EFFECT OF ETHANOL ON THE KINETICS OF CHLORAL HYDRATE METABOLISM IN MICE.
 Fed. Proc. (Bethesda), 26: 568 (0 ref.), 1967.
 E – abst. – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo –
 metab. proc. – sed., hypnot. – *CAAAL-0 B-0935.

Chloral hydrate (CH) was given ip to female mice; group 1 received 500 mg/kg, and group 2 the same CH dose plus an equimolar amount of ethanol. Mice were killed at various times following administration, frozen in liquid nitrogen, homogenized with sulphosalicylic acid, and assayed for chloral hydrate and its metabolites. Coadministration of ethanol resulted in an increased metabolism of CH. In group 1, the rate constants, in min^{-1} , for CH disappearance and for the formation of trichloroethanol and trichloroacetic acid were .058, .032, and .0064, respectively, while the corresponding constants in group 2 were .075, .059, and .0059, respectively. The rate constant for the formation of urochloralic acid was unaffected by ethanol. It is concluded that the observed differences in rate constants account in part for the potentiation of effects.

474. Gessner, P. K., and Cabana, B. E.
CHLORAL ALCOHOLATE: REEVALUATION OF ITS ROLE IN THE INTERACTION BETWEEN THE HYPNOTIC EFFECTS OF CHLORAL HYDRATE AND ETHANOL.
 J. Pharmacol. Exp. Ther. (Baltimore), 156(3): 602-605 (18 ref.), 1967.
 E – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose
 resp. – absorp., distrib., stor. – liver, kidney – metab. proc. – sed., hypnot. – *CAAAL-12551-B2
 B-0301.

In mice, the hypnotic properties of aqueous sol of chloral alcoholate were compared with those of chloral hydrate. Chloral alcoholate, both on a weight and a molar basis, was found to be a significantly more potent hypnotic than chloral hydrate. The potency ratios are presented in a table. The acute toxicity of the two agents was compared by determination of the 24-hr non-aggregated LD_{50} 's.

475. Gessner, P. K., and Cabana, B. E.

A STUDY OF THE INTERACTION OF THE HYPNOTIC EFFECTS AND OF THE TOXIC EFFECTS OF CHLORAL HYDRATE AND ETHANOL.

J. Pharmacol. Exp. Ther. (Baltimore), 174(2): 247-259 (30 ref.),

1970.

E – exp. comp. – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – sed., hypnot. – *CAAAL-0 B-0571.

The synergism between ethanol (E) and chloral hydrate (CH) was investigated in male Swiss Webster mice. The ED₅₀'s (using loss of righting reflex as criterion) were determined for E and CH by calculating a dose-response curve for various doses of each agent administered in combination with a fixed dose of the other drug, and by the plotting of isobolograms (Loewe's method). The results indicated a significant potentiation or supra-addition of hypnotic effects for mixtures in which the agents were present in a wt ratio equal to or greater than 1:7.2, and simple addition of effects for mixtures with smaller wt ratios. To investigate the possibility that trichloroethanol (T), an active metabolite of CH, is the basis of potentiation of E by CH (since T has been observed to inhibit in vitro E oxidation), the effects of T plus E were determined and found to be only additive (thus indicating a direct interaction of CH and E). The toxic combined effects of CH and E were investigated, using determination of 24-hr non-aggregated LD₅₀'s. Mixtures with wt ratios of 3.6:1 (1:1 M ratio) showed significant potentiation; mixtures with wt ratios of 1:5, 1:10, and 1:15 revealed partial but significant antagonism; and mixtures with ratios of 1:1, 1:1.14, 1:3, and 1:30 showed simple addition of toxic effects.

476. Gettler, D. T., and Allbritten, F. F.

EFFECT OF ALCOHOL INTOXICATION ON THE RESPIRATORY EXCHANGE AND MORTALITY RATE ASSOCIATED WITH ACUTE HEMORRHAGE IN ANESTHETIZED DOGS.

Ann. Surg. (Philadelphia), 158(2): 151-158 (17 ref.),

1963.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – acid-base, blood pH, elect. – respir. – barbiturates – *CAAAL-0 A-1396.

Tests were conducted on 4 groups of adult splenectomized mongrel dogs (7.8-16.8 kg). Groups 1 (control), 3, and 4 were given 30 mg/kg sodium pentobarbital iv, plus 3.0 g/kg ethanol as a 25% aqueous sol by gastric tube, while group 2 (control) received pentobarbital (above dose) plus a vol of water equal to that of the alcohol dose. In groups 2 and 3, acute hemorrhage was induced by removal of 35% of the measured blood vol from a femoral artery, and, in group 4, hemorrhage was followed by room air positive pressure assisted respiration at a rate of 10 l/min for 1 hr. Alcohol, pH, oxygen, and carbon dioxide contents of the blood were determined. A mortality rate of 30% resulted in intoxicated animals subjected to acute hemorrhage, whether or not artificial respiration was applied. Without respiratory depression produced by alcohol, there was an increase in respiratory minute vol, and metabolic acidosis was corrected by compensatory respiratory alkalosis. In intoxicated animals, acidosis was not corrected, and arterial blood pH remained low, although artificial respiration restored pH to near pre-hemorrhagic levels. In all control animals, mean blood pressure returned to 70 mm Hg or higher after 3 hr, whereas, after the same time, only 1/2 of the intoxicated animals had a blood pressure which reached this level; however, all surviving intoxicated dogs given ventilatory assistance reached the 70 mm level.

477. Ghosh, J. J., and Quastel, J. H.

NARCOTICS AND BRAIN RESPIRATION.

Nature (London), 174: 28-31 (17 ref.),

1954.

E – exp. comp. – DC (decrease) – mammals – in vitro – metab. proc. – anticonvulsants – elect., water-bal. agents – hormones, hormone antag. – *CAAAL-7698-B2 A-0727.

Brain cortex slices from rats and guinea pigs were allowed to respire at 37°C in oxygen, in a medium containing sodium chloride, potassium chloride, calcium chloride, magnesium sulfate, potassium biphosphate, sodium biphosphate, and, usually, glucose. A narcotic sol was tipped into the medium when the respiration became constant. The addition of ethanol under normal (unstimulated) conditions increased the rate of respiration, the increase diminishing with rising ethanol concentrations. In presence of KCl, however, even the lowest concentration tested produced an inhibition of respiration, the inhibition increasing with increasing ethanol concentration. Further effects are discussed in detail.

478. Giese, E.
UEBER DIE VERÄNDERUNGEN DER NERVENZELLEN BEI DER AKUTEN
VERGIFTUNG DURCH ALKOHOL UND FUSELÖL. [Changes in nerve cells after acute
poisoning with ethanol and fusel oil].
Monatschrift für Psychiatrie und Neurologie (Basel), 11: 237 (0 ref.), 1902.
G – exp. comp. – congen. stud. – mammals – acute admin. – in vivo – CNS – nerv. syst. – *CAAAL-0
A-1333.

Ethanol and fusel oil, separately or in combination in various concentrations, were administered po or by injection into the saphena vein of dogs. Experiments were divided into 3 groups: (1), basic experiments on dogs in which samples of the sulci cruciati, olfactory lobes, cerebellum, medulla oblongata, spinal cord, intervertebral ganglia, and coeliac were taken, during complete narcosis; (2) experiments on dogs in which the same samples were taken 3 times—before poisoning, during narcosis, and after death; (3) experiments on young dogs, cats, and dog embryos. The results showed that poisoning by fusel oil, especially in combination with ethanol, markedly affected the nerve cells, much more than did ethanol alone. Nerve cells were affected in 2 ways—1), there was coagulation necrosis, and 2), cells became bloated, the colourless substance became intensely coloured, and Nissl's bodies became bloated, fell apart, and disappeared. The nucleus withered, lost its shape, and became intensely coloured. Normal cells were found among the changed ones, the percentage of normal ones being highest in the pure ethanol group.

479. Gilger, A. P., Potts, A. M., and Johnson, L. V.
STUDIES ON THE VISUAL TOXICITY OF METHANOL. II. THE EFFECT OF
PARENTERALLY ADMINISTERED SUBSTANCES ON THE SYSTEMIC TOXICITY OF
METHYL ALCOHOL.
Amer. J. Ophthal. (Chicago), 35(5, part 2): 113-123 (28 ref.), 1952.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp.
– CNS – metab. proc. – alcohols – *CAAAL-6226-B2 A-0728.

The LD₅₀ for methanol and formaldehyde was established for mice. In equimolar amounts, formaldehyde was found to be 145 times as lethal as methanol. Administration of disulfiram lowered the LD₅₀ for methanol from 10.5 to 5.5 g/kg. Ethanol was given every 4 hr, beginning just before the single injection of methanol. Three different doses of ethanol were tried: 2.0 g/kg changed the LD₅₀ for methanol from 10.5 to 5.5 g/kg; 0.674 g/kg, followed by 0.404 g/kg, raised the mortality of mice from 40% to 100%; and a dose of 0.17 g/kg every 4 hr raised the mortality by 40%. The findings indicate that ethanol therapy for the condition should be discontinued, pending further investigation.

480. Gilger, A. P., Potts, A. M., and Farkas, I. S.
STUDIES ON THE VISUAL TOXICITY OF METHANOL. IX. THE EFFECT OF
ETHANOL ON METHANOL POISONING IN THE RHESUS MONKEY.
Amer. J. Ophthal. (Chicago), 42(10, part 2): 244-252 (18 ref.), 1956.
E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – acid-base, blood
pH, elect. – blood comp., sites, lymph – metab. proc. – senses – alcohols – *CAAAL-0 A-0729.

Experiments were carried out on 5 male rhesus monkeys to show the antidotal properties of ethanol in methanol poisoning. The monkeys were first given large doses of methanol (4.0-6.2 g/kg 20% methanol) by stomach tube (doses presumed to be lethal), immediately followed by small repeated doses of ethanol. All 5 monkeys survived, and showed no signs of eye damage. After 3 weeks recuperation, the same single dose of methanol alone was given. 4 of the 5 monkeys died quickly, and the fifth died 18 days afterwards. It is concluded that ethanol is an effective antidote for methanol poisoning in primates, due to the apparently unique course of the poisoning in this group; previous studies have shown that ethanol is ineffective in poisoned non-primates (Amer. J. Ophthal. (Chicago), 35(5, part 2): 113-123, 1952).

481. Gilger, A. P., Farkas, I. S., and Potts, A. M.
STUDIES ON THE VISUAL TOXICITY OF METHANOL. X. FURTHER OBSERVATIONS ON THE ETHANOL THERAPY OF ACUTE METHANOL POISONING IN MONKEYS.
 Amer. J. Ophthal. (Chicago), 48(1, part 3): 153-161 (8 ref.), 1959.
 E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – blood lev. – acid-base, blood pH, elect. – blood comp., sites, lymph – CNS – metab. proc. – nerv. syst. – senses – alcohols – *CAAAL-9408-A2 A-0730.

Single doses of 6 g of methanol/kg po were given to 5 monkeys. 80% of the untreated monkeys died 17 to 38 hr after such a dose. When ethanol was administered in doses between 0.5 and 1.0 g/kg every 4 hr, starting within 8 to 12 hr after the methanol, the monkeys survived. If the administration of ethanol was started 12 to 20 hr after the methanol, death occurred 40 to 136 hr after the methanol.

482. Gilger, A. P.
THE TREATMENT OF METHANOL POISONING; A REVIEW.
 J. Amer. Med. Wom. Ass. (Nashville), 16(5): 379-382 (22 ref.), 1961.
 E – general – DC (antidotal) – humans – acid-base, blood pH, elect. – blood comp., sites, lymph – CNS – metab. proc. – nerv. syst. – respir. – senses – alcohols – *CAAAL-10342-N38 A-0731.

A review of the treatment of methanol poisoning is presented. Death from acidosis can often be prevented by alkalinization. Death from acidosis, and death or blindness from nervous system involvement, can be prevented by ethanol therapy, but only when given early after poisoning. The recommended ethanol dosage (10-20% po or 5% iv) for all patients seen within 2 days after methanol poisoning is 0.75 g/kg ethanol at once, followed by 0.5 g/kg every 4 hr for about 3 days. Experimental searching for more effective antagonists to nervous system damage is continuing.

483. Girard
DES PROPRIÉTÉS MÉDICALES DE L'ALCALI VOLATIL FLUOR (AMMONIAQUE LIQUIDE) EN GÉNÉRAL, ET PARTICULIÈREMENT DANS L'IVRESSE. [Some medical properties of liquid volatile alkali (liquid ammonia) in general, and particularly in intoxication].
 Journal Générale de Médecine, de Chirurgie, et de Pharmacie; ou, Recueil Périodique de la Société de Médecine de Paris (Paris), 73: 166-178 (3 ref.), 1820.
 F – general – case hist. – DC (antidotal) – humans – CNS – stimulants – *CAAAL-0 A-0732.

Cases are presented in which ammonia was employed therapeutically, with most satisfactory results, for various maladies, including inebriety. One case involved a young woman who, after ingestion of white wine, became intoxicated. Treatment with 6 drops of ammonia in half a glass of sweetened water restored the woman's balance instantly. Another case of intoxication (3-4 glasses of wine), resulting in physical and mental disturbances, responded within minutes to a few swallows of a mixture of 20 drops of ammonia in a glass of water. Afterwards, the patient passed a restful night with no untoward after-effects. Other cases with satisfactory outcome are mentioned.

484. Girdlestone, T. M.
 THE INTRAVENOUS INJECTION OF AMMONIA IN ACUTE ALCOHOLISM.
 Australian Medical Journal (Melbourne), nsv. 2: 141-143 (3 ref.), 1880.
 E – general – case hist. – DC (antidotal) – drug-dep. humans – cardiovasc. – CNS – stimulants –
 *CAAAL-0 A-0733.

Several reported cases of alcoholic poisoning treated by ammonia are discussed. The author contends that a number of cases of snake-poisoning reported as recoveries through iv ammonia injections, especially those of persons in a profound coma who had been immediately resuscitated, were in reality cases of alcoholic poisoning. In fact, he is convinced that the coma of snake-poisoning cannot be relieved by ammonia.

485. Glass, F., Gossow, H., and Mallach, H. J.
 BEOBACHTUNGEN UND UNTERSUCHUNGEN ÜBER DIE GEMEINSAME WIRKUNG
 VON ALKOHOL UND ISONICOTINSÄUREHYDRAZID. [Observations and investigations of
 the combined effect of alcohol and isonicotinic acid hydrazide].
 Arzneimittelforschung (Aulendorf), 14(11): 1203-1208 (29 ref.), 1964.
 G – ES – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals
 – acute admin. – in vivo – dose resp. – blood lev. – anti-infectants – *CAAAL-0 A-0734.

The median lethal dose of isoniazid (INH) in mice, following po administration, yielded a value of 176 mg/kg. Survival times were reduced with increased doses, and approached the value 0 with a dose of 331 mg/kg. Simultaneous administration of alcohol and INH increased the lethal effect. Small INH doses lowered the toxicity of alcohol. Small alcohol doses completely neutralized the lethal effect of LD₅₀-INH doses. Animal experiments are compared with human case material. 7 patients who consumed alcohol while on INH therapy showed very pronounced signs of intoxication; the average blood alcohol concentration was 2°/oo, corresponding (in an 80 kg person) to a consumption of 8 glasses of beer and 8 drinks of spirits (containing 112 g anhydrous alcohol).

486. Glass, F., Mallach, H. J., and Simsch, A.
 BEOBACHTUNGEN UND UNTERSUCHUNGEN ÜBER DIE GEMEINSAME WIRKUNG
 VON ALKOHOL UND D-CYCLOSERIN. [Observations and investigations concerning the
 combined effects of alcohol and D-cycloserine].
 Arzneimittelforschung (Aulendorf), 15(6): 684-688 (30 ref.), 1965.
 G – ES – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals
 – acute admin. – in vivo – dose resp. – blood lev. – cardiovasc. – anti-infectants – *CAAAL-0
 B-0302.

The median lethal dose of D-cycloserine (CS) in mice, following oral administration, yielded a value of 11980 mg/kg. Survival time diminished with increasing doses. Simultaneous administration of alcohol and CS increased mortality. Small CS doses enhanced alcohol effects, whereas small doses of alcohol reduced mortality of CS. Animal experiments are compared with human case material. Two patients who consumed alcohol under CS therapy had blood alcohol concentrations ranging from 1.17 to 2.2°/oo. Both had excessive reactions to alcohol, indicating a qualitative enhancement of the narcotic effect of alcohol.

487. Glass, F., and Mallach, H. J.
 TIEREXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE ALKOHOLWIRKUNG
 NACH LÄNGERER BELASTUNG MIT ISONICOTINSÄUREHYDRAZID. [Experimental
 animal investigations into the effect of alcohol after sustained loading with isonicotinic acid
 hydrazide].
 Arzneimittelforschung (Aulendorf), 15(9): 1069-1070 (1 ref.), 1965.

G – ES – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – dose resp. – anti-infectants – *CAAAL-1178-B2 B-0303.

The results obtained in 164 individual tests on mice, designed to clarify the effect of alcohol during sustained isonicotinic acid hydrazide (INH) toxicosis, differed in no way from the findings reported earlier (Arzneimittelforschung (Aulendorf), 14(11): 1203-1208, 1964), concerning the combined effects of alcohol and INH. Any particular influence of INH on body wt was, in general, not found.

488. Glass, F., and Mallach, H. J.

BEOBACHTUNGEN UND UNTERSUCHUNGEN ÜBER DIE GEMEINSAME WIRKUNG VON ALKOHOL UND α -PHENYL- α -AETHYL-GLUTARSÄUREIMID (GLUTETHIMID). [Observations and investigations on the joint action of alcohol and α -ethyl- α -phenylglutaric acid amide (glutethimide)].

Arzneimittelforschung (Aulendorf), 16(4): 528-532 (7 ref.), 1966.

G – ES – exp. cont. – DC (decrease) – DC (supra-add. incr.) – humans – mammals – acute admin. – in vivo – dose resp. – blood lev. – sed., hypnot. – *CAAAL-0 B-0304.

The LD₅₀ of glutethimide after po administration to mice was established at 514 mg/kg. Alcohol and glutethimide, in doses corresponding to the LD₅₀'s, increased the mortality when given simultaneously. Even small doses of the drug increased the alcohol toxicity, compared to the LD₅₀, by almost 100%; in contrast to this, small alcohol doses considerably reduced the mortality of glutethimide. Evaluation of 11 cases of suicide with glutethimide showed a lethal dose of 175 mg/kg for humans, with a probable survival time of 33 hr; of these cases, only 1 had ingested alcohol (blood alcohol concentration of 0.71°/oo—he had taken a dose of 198 mg/kg glutethimide, and survived about 72 hr.

489. Gold, H., and Travell, J.

ETHYL ALCOHOL AND STRYCHNINE ANTAGONISM.

J. Pharmacol. Exp. Ther. (Baltimore), 52: 30-53 (21 ref.), 1934.

E – exp. cont. – exp. comp. – DC (decrease) – mammals – other org. – acute admin. – in vivo – dose resp. – species or sex diff. – cardiovasc. – CNS – respir. – skel., muscle, skin – stimulants – *CAAAL-0 A-0735.

Experiments were conducted on cats, dogs, rabbits, and frogs. It was found that the cat and dog can be protected against as much as 9 times the minimum lethal dose of strychnine by alcohol. Alcohol was somewhat less effective in the rabbit, and marked antagonism was found in the frog. In the cat, the maximum effect of 1-2 cc alcohol was required to protect against about 0.2 mg of strychnine. The cats recovered from strychnine at a rate equivalent to the excretion of approximately 0.05-0.1 mg/kg/hr, and from alcohol in large doses at a rate equivalent to 0.25-0.5 cc/kg/hr. The maximum dose of strychnine which the animals survived when treated by alcohol was only slightly greater after sc than after iv injections; large doses of alcohol po, followed by small iv doses throughout the course of the poisoning whenever excessive hyperexcitability or convulsions occurred, gave the most effective protection.

490. Gold, H., and Travell, J.

STRYCHNINE IN POISONING BY ALCOHOL.

J. Pharmacol. Exp. Ther. (Baltimore), 52: 345-354 (5 ref.), 1934..

E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – cardiovasc. – CNS – metab. proc. – respir. – skel., muscle, skin – stimulants – *CAAAL-0 A-0736.

The effect of strychnine was investigated in cats and dogs under the influence of alcohol, with respect to respiration, higher centers (drowsiness, ataxia, narcosis), spinal cord, muscular tone, and rate of

recovery from the effects of alcohol. Antagonism between alcohol and strychnine did not extend to all the actions of the two drugs in the same degree. Strychnine reversed the alcohol depression of the higher centers only to a slight degree. The mutual antagonism between the two drugs was most pronounced with respect to the action on the spinal cord (reflex hyperexcitability and tetanus).

491. Gold-Aubert, P., and Lacroix, J.
 DE LA POTENTIALISATION DES EFFETS DE L'ALCOOL PAR QUELQUES
 PSYCHOTROPES. [The potentiation of the effects of alcohol by some psychotropic drugs].
 Médecine et Hygiène (Geneva), 918: 855-860 (17 ref.), 1970.
 F – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp.
 – mot. perform. – psychol. perform. – CNS – barbiturates – tranquilizers – *CAAAL-0 B-0518.

Combined effects of psychoactive drugs with alcohol were tested in mice. Evasion, curiosity, motility, circular tunnel, and equilibrium (turning rod) tests were given to mice after administration of various doses of chlorpromazine, phenobarbital, oxazepam, or atrium, with or without 4 g/kg alcohol po. The psychosedatives, chlorpromazine, phenobarbital, and oxazepam produced the expected synergism with alcohol; atrium, however, produced none. With alcohol, atrium had a psychoanaleptic, rather than a psychosedative, effect, even in doses of up to 200 mg/kg. The observed progressive psychostimulant effect of atrium in weak doses, and the psychosedative effect in strong doses, might be profitably used in alcohol disintoxication treatment.

492. Goldberg, L.
 ALCOHOL, TRANQUILIZERS AND HANGOVER.
 Quart. J. Stud. Alcohol (New Haven), Suppl. 1: 37-56 (16 ref.), 1961.
 E – exp. cont. – exp. comp. – congen. stud. – DC (add., infra-add., unspec. incr.) – humans – acute
 admin. – in vivo – blood lev. – mot. perform. – CNS – nerv. syst. – tranquilizers – *CAAAL-9663-D1
 A-0737.

In 80 healthy men, the acute effects of alcohol po and tranquilizers—meprobamate (400 + 400 mg), phenoglycodol (300 + 300 mg), buclizine (50 + 50 mg), hydroxyzine (25 + 25 mg), and chlorpromazine (10 + 10 mg), all administered po—on the CNS were studied immediately after alcohol intake and during hangover, in a total of 224 experiments. Electrooculography, electroencephalography, and the standing steadiness test were used. Subjective symptoms (sleepiness and fatigue) were much more severe when drugs and alcohol were given together, than after alcohol alone, and standing steadiness was also more impaired. Many other aspects of the experiments are presented in detail.

493. Goldberg, L.
 EFFECTS AND AFTER-EFFECTS OF ALCOHOL, TRANQUILLIZERS AND FATIGUE
 ON OCULAR PHENOMENA.
 In: Havard, J.D.J., ed. *Alcohol and Road Traffic*. Proceedings of the Third International Conference
 on Alcohol and Road Traffic at London, September 3-7, 1962. London: British Medical Association,
 pp. 123-135 (20 ref.), 1963.
 E – exp. cont. – exp. comp. – presentation – congen. stud. – DC (add., infra-add., unspec. incr.) –
 humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform.
 – absorp., distrib., stor. – acid-base, blood pH, elect. – CNS – nerv. syst. – analg., antipyret. –
 tranquilizers – *CAAAL-0 A-0738.

A total of 250 human subjects, all moderate drinkers, took part in the experiments. The methods used in this study included electrooculography (EOG) to record positional alcohol nystagmus (PAN) and roving ocular movements (ROM). The objective and subjective effects and after-effects of alcohol consumption were recorded for 5-24 hours, and were especially noticeable on PAN, ROM, standing steadiness, and fatigue. The addition of tranquilizers modified the subjective and objective effects and

after-effects in various quantitatively different ways. The combinations of alcohol and buclizine, chlorpromazine, hydroxyzine, meprobamate, phenoglycodole, chlordiazepoxide, including codeine, and acetylsalicylic acid were tested. At a certain blood alcohol level, the subject was considerably more intoxicated after having taken alcohol plus a tranquilizer, than after alcohol plus placebo.

494. Goldberg, L.

ALCOHOL, CNS-ACTIVE DRUGS AND DRIVING SKILL.

Fourth International Congress of the International Federation for Hygiene and Preventive Medicine, Vienna, Austria, May 24-26, 3 pp. (0 ref.), 1965.

E – exp. cont. – exp. comp. – presentation – DC (decrease) – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – CNS – nerv. syst. – tranquilizers – *CAAAL-0 B-0305.

A preliminary report is given of a study made on the effect of alcohol, CNS-active agents, and combinations of alcohol and drugs on driving skill. Field and laboratory experiments were carried out. Tests included rating of subjective mood estimates, objective performance, and physiological functions. The drugs used had no influence on the blood alcohol curve. All tranquilizers tested, except chlordiazepoxide, brought about a synergistic effect with alcohol. Chlordiazepoxide had an antagonistic effect and decreased impairment, thus improving performance.

495. Goldberg, L.

INTERACTION BETWEEN ALCOHOL AND TRANQUILIZING AGENTS.

In: Harger, Rolla N., ed. *Alcohol and Traffic Safety*. Proceedings of the Fourth International Conference on Alcohol and Traffic Safety at Indiana University, December 6-10, 1965. Bloomington, Indiana: Indiana University Press, pp. 235-244 (14 ref.), 1966.

E – exp. cont. – exp. comp. – presentation – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – CNS – metab. proc. – nerv. syst. – amphetamines – autocoids – gastrointest. agents – sed., hypnot. – tranquilizers – *CAAAL-0 B-0306.

A total of 540 experiments were carried out on 160 healthy humans to assess some behavioural and physiological effects of alcohol ingestion. The combinations of alcohol (in the form of whiskey, 0.33-0.66 g alcohol/kg) and the following 11 CNS-active drugs po were studied: amphetamine (10 mg), buclizine (50 mg), chlorcyclizine (50 or 100 mg), chlordiazepoxide (20 mg), chlorpromazine (10 mg), hydroxyzine (25 mg), meclizine (25 or 50 mg), meprobamate (400, 500, or 800 mg), phenoglycodole (300 mg), promethazine (25 mg), and tripeleennamine (50 mg). None of the drugs changed the course of the blood alcohol curve in any essential way, the effect of the interaction varying with the type of drug and the phase of alcohol metabolism studied. Meprobamate acted synergistically in the acute alcohol phase and reduced some of the after-effects in the post-alcohol phase; chlordiazepoxide acted antagonistically in all phases. This study is published in expanded form in *Psychosom. Med.* (New York), 28(4, part 2): 570-595, 1966.

496. Goldberg, L.

BEHAVIORAL AND PHYSIOLOGICAL EFFECTS OF ALCOHOL ON MAN.

Psychosom. Med. (New York), 28(4, part 2): 570-595 (23 ref.), 1966.

E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – CNS – metab. proc. – nerv. syst. – autocoids – gastrointest. agents – sed., hypnot. – tranquilizers – amphetamines – *CAAAL-0 B-0307.

A total of 540 experiments were carried out on 160 healthy humans to assess some behavioural and physiological effects of alcohol ingestion. The combinations of alcohol (in the form of whiskey,

0.33-0.66 g alcohol/kg) and the following 11 CNS-active drugs po were studied: amphetamine (10 mg), buclizine (50 mg), chlorcyclizine (50 or 100 mg), chlordiazepoxide (20 mg), chlorpromazine (10 mg), hydroxyzine (25 mg), meclizine (25 or 50 mg), meprobamate (400, 500, or 800 mg), phenaglycodole (300 mg), promethazine (25 mg), and tripeleennamine (50 mg). None of the drugs changed the course of the blood alcohol curve in any essential way, the effect of the interaction varying with the type of drug and the phase of alcohol metabolism studied. Meprobamate acted synergistically in the acute alcohol phase, and reduced some of the after-effects in the post-alcohol phase; chlordiazepoxide acted antagonistically in all phases. This study is also reported in condensed form in: Harger, Rolla N., ed., *Alcohol and Traffic Safety: Proceedings of the Fourth International Conference on Alcohol and Traffic Safety at Indiana University, December 6-10, 1965* (Bloomington, Indiana, 1966), pp. 235-244.

497. Goldberg, L., and Rydberg, U.
INHIBITION OF ETHANOL METABOLISM *IN VIVO* BY ADMINISTRATION OF PYRAZOLE.
 Biochem. Pharmacol. (London), 18: 1749-1762 (17 ref.), 1969.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – in vitro – dose resp. – blood lev. – mot. perform. – absorp., distrib., stor. – blood comp., sites, lymph – CNS – liver, kidney – metab. proc. – *CAAAL-14274 B-0519.

Ethanol (12% w/v sol in distilled water) was injected ip into rats in a dose of 32.6 m-mole/kg (1.5 g/kg). Pyrazole (2% w/v sol in distilled water) was similarly injected ip, either 15 min before or 120 min after the ethanol administration, in doses of 0.07-8.82 m-mole/kg. The blood level of ethanol was determined at 30 min, and later at 60 min intervals after ethanol administration until it reached zero. Doses of 0.07-1.76 m-mole/kg pyrazole, administered 120 min after ethanol, caused a 35-83% decrease in ethanol metabolism. 8.82 m-mole/kg pyrazole, administered 15 min prior to ethanol, extended the elimination time from 235 min up to 2100 min, and decreased the rate of elimination from 45 mg ethanol/kg blood per min to 0.8. At all levels of pyrazole used, an increase in the dose of ethanol would decrease the degree of inhibition. Pyrazole in combination with ethanol produced a greater degree of impairment in the animals than did the ethanol alone. It is concluded that pyrazole competitively inhibits ethanol metabolism in vivo.

498. Goldstein, L., and Beck, R. A.
AMPLITUDE ANALYSIS OF THE ELECTROENCEPHALOGRAM: REVIEW OF THE INFORMATION OBTAINED WITH THE INTEGRATIVE METHOD.
 Int. Rev. Neurobiol. (New York), 8: 265-312 (90 ref.), 1965.
 E – SEC – review – DC (add., infra-add., unspec. incr.) – mammals – blood lev. – CNS – amphetamines – hallucinogens – hormones, hormone antag. – sed., hypnot. – stimulants – *CAAAL-0 B-0308.

The article reviews the knowledge that can be obtained with the amplitude analysis of the electroencephalogram. Methods of measurement and analysis of measurement are discussed in detail. Analysis of changes produced by every category of centrally-active drugs is reviewed. Some published experimental work concerning the effects of various stimulants on rabbits pretreated with ethanol is mentioned briefly.

499. Goreczky, L.
A VÉR ALKOHOLTARTALMÁNAK BEFOLYÁSOLHATÓSÁGARÓL. [Modification of the concentration of alcohol in the blood].
 Orvostképzés (Budapest), 32: 391-396 (17 ref.), 1942.
 H – review – DC (decrease) – mammals – blood lev. – CNS – hormones, hormone antag. – stimulants – *CAAAL-0 A-0739.

A review of literature is given in which the author makes reference to the influence of some drugs on inebriety. It is pointed out that, while caffeine has no direct effect on the blood alcohol level, it does act on the whole nervous system. In previously published experiments with rabbits, 2 IU/kg insulin decreased the blood alcohol level. However, these results could not be reproduced in diabetic patients. In other experiments, results of 40 IU insulin with moderate quantities of alcohol varied from subject to subject. Although insulin was found to decrease the blood alcohol level in some subjects, its use as a sobering agent is dangerous, because it may induce low blood sugar shock or coma.

500. Göres, E.

EINIGE PHARMAKOLOGISCHE PROBLEME DER ÄTHANOLWIRKUNG. 1. TEIL; 2. TEIL. [Some pharmacological problems with respect to the effects of ethanol. Part 1; Part 2]. Pharmazie (Berlin), 19(7 and 8): 433-448, and 489-507 (389 ref.), 1964.
G – review – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – DC (sensit.) – humans – mammals – absorp., distrib., stor. – cardiovasc. – CNS – metab. proc. – analg., antipyret. – anti-infectants – elect., water-bal. agents – hormones, hormone antag. – indust. intox. – miscellaneous – sed., hypnot. – unclass. ther. agents – *CAAAL-0
A-0740.

Reviewed in detail is the literature on the pharmacological problems of the effects of endogenous and exogenous ethanol in man and animals. Discussed are acute intoxication and acute lethal doses, chronic intoxication, and the effects of alcohol on metabolism and on all human organs and systems. One section deals with the interaction of alcohol and other drugs, and the antagonistic or synergistic effects of different drug groups are discussed. The groups include insulin, sedatives and analgetics, opiates, ataractics, oral antidiabetics, ethanol sensitizers, and industrial poisons.

501. Goulston, K., and Cooke, A. R.

ALCOHOL, ASPIRIN, AND GASTROINTESTINAL BLEEDING. Brit. Med. J. (London), 4(5632): 664-665 (5 ref.), 1968.
E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – acid-base, blood pH, elect. – blood comp., sites, lymph – G.I. tract – analg., antipyret. – *CAAAL-0
B-0309.

In this clinical study, faecal blood loss was measured in 20 healthy male subjects by means of a chromium-51-labelled red blood cell technique. In response to alcohol plus aspirin, the faecal blood loss in 11 of the 13 subjects was greater than that during the aspirin period. Mean daily faecal blood loss in response to aspirin plus alcohol (5.3 ± 0.5 ml) was significantly greater than that in response to aspirin alone. In seven others, alcohol alone did not cause gastrointestinal bleeding. These findings suggest that alcohol may accentuate gastrointestinal blood loss associated with unbuffered aspirin ingestion.

502. Grabill, F. J., Hockmuth, L., and Tuohy, E. B.

THE INTRAVENOUS USE OF ALCOHOL IN SURGERY; POSTOPERATIVE USE OF 7.5 PER CENT ETHYL ALCOHOL, 5 PER CENT PROTEIN HYDROLYSATE AND 5 PER CENT DEXTROSE IN 1000 CC OF WATER. Anesth. Analg. (Cleveland), 29: 211-216 (13 ref.), 1950.
E – SEC – review – DC (add., infra-add., unspec. incr.) – humans – analg., antipyret. – *CAAAL-0
A-0741.

Alcohol (7.5%) when given iv was found, in 305 patients, to be a safe analgesic and sedative that can be used during conduction or general anesthesia. Preliminary reports indicate that the 7.5% alcohol, 5% protein hydrolysate, and 5% dextrose sol contains sufficient calories to provide complete nutritional requirements for the average postoperative patient. The combination of 7.5% alcohol and 0.1% procaine hydrochloride was found materially to increase the analgesic properties.

503. Grady, R. W., and Rich, A. L.
 CLINICAL EVALUATION OF PARENTERAL HYDROXYZINE FOR PREOPERATIVE MEDICATION.
 Southern. Med. J. (Birmingham), 54: 766-768 (11 ref.), 1961.
 E – SEC – exp. – DC (unchanged) – drug-dep. humans – acute admin. – in vivo – CNS – G.I. tract – tranquilizers – *CAAAL-9330-E7 A-0742.

In the pre-operative preparation of 400 cases, 25 to 100 mg of hydroxyzine was given parenterally in combination with atropine and 1/2 of the calculated therapeutic dose of pethidine. In the preparation of patients suffering from acute alcoholism, parenteral hydroxyzine in 50-100 mg doses produced a quieting effect without the depression observed with other drugs. All 10 patients in this condition became cooperative, and none had postoperative nausea or emesis in the immediate postoperative period.

504. Graf, O.
 INCREASE OF EFFICIENCY BY MEANS OF PHARMACEUTICS (STIMULANTS).
 In: *German Aviation Medicine: World War II. II.* Washington: Dept. of the Air Force, pp. 1080-1103 (28 ref.), 1950.
 E – SEC – exp. cont. – exp. comp. – review – DC (decrease) – humans – CNS – amphetamines – stimulants – *CAAAL-0 A-0743.

The effects of various stimulants on mental and motor performance with respect to flying efficiency are discussed. The results of an experiment are given in which it was attempted to neutralize the negative effect of ethanol on psychic functions by combining it with stimulants. 2 male test subjects received 30 g alcohol, and 2 female subjects 20 g alcohol, in combination with 0.05 g caffeine/10 g alcohol, 3 mg pervitin/10 g alcohol, or 0.025 g caffeine plus 0.05 g cardiazol/10 g alcohol. Performance tests given before and after administration showed that all stimulants counteracted the alcohol effect. Pervitin had the strongest and most prolonged effect; not a single subject showed the usual immediate effect of alcohol, and euphoria and confusion rapidly faded. The caffeine-cardiazol combination showed a similar effect for 1 hr, but did not last as long as pervitin, and its later course was similar to that of caffeine. It is concluded that it is possible to neutralize even a marked alcohol effect, but, in practice, this method should be employed only as an emergency aid.

505. Graham, J. D. P.
 ETHANOL AND THE ABSORPTION OF BARBITURATE.
 Toxic. Appl. Pharmacol. (New York), 2: 14-22 (6 ref.), 1960.
 E – exp. cont. – congen. stud. – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – mammals – acute admin. – in vivo – dose resp. – blood lev. – other drug lev. – absorp., distrib., stor. – CNS – barbiturates – *CAAAL-9649-D2 A-0744.

The interaction of barbiturates and ethanol was investigated by examination of case records of humans, and by experiments in mice and rats. Records concerning 11 pairs of matched patients suffering from acute barbiturate poisoning, 1 of each pair having also consumed alcohol, were studied. It was concluded that the severity of symptoms was greater in alcoholics and in drinkers of spirits than in drinkers of ale, but blood levels were not significantly altered by alcoholism. In mice given 25%, 50% or 75% of the LD₅₀ of 40% alcohol plus the LD₅₀ of pentobarbitone sodium, an additive effect was found. In rats given 10 ml/kg water, 10 ml/kg 20% ethanol:water, or 5 ml/kg 40% ethanol:water po, plus 80 mg/kg pentobarbitone sodium, there was no difference in brain levels of barbiturates. In rats given 10 ml/kg of water or of 20% ethanol:water po, plus 80 mg/kg pentobarbitone sodium, blood levels were just significantly lower with the alcohol. The experimental results show an additive synergism.

506. Graham, R. C. B., Lu, F. C., and Allmark, M. G.
 COMBINED EFFECT OF TRANQUILIZING DRUGS AND ALCOHOL ON RATS.
 Fed. Proc. (Bethesda), 16: 302 (1 ref.), 1957.
 E – abst. – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals
 – acute admin. – in vivo – dose resp. – CNS – sed., hypnot. – tranquilizers – *CAAAL-0
 A-0745.

The sloping screen technique was used to examine the effect of 50% alcohol ip or po on rats pretreated with glutethimide, methyprylone, meprobamate, promazine, captodiamine, N-methylpiperidyl-(3)-methylphenothiazine (pacatal), ethinamate, hydroxyzine, ethyl trichloramate, and azacyclonol. These drugs, except hydroxyzine, azacyclonol, and possibly ethinamate, enhanced the alcohol effect. Azacyclonol antagonized the alcohol effect. In another experiment, the loss of righting reflex was the criterion; promazine enhanced the alcohol effect for as long as 10 hr after alcohol. The results suggest that the combined effect of the tranquilizers and alcohol is additive rather than supra-additive.

507. Gray, I.
 CARBON TETRACHLORIDE POISONING: REPORT OF SEVEN CASES WITH TWO DEATHS.
 New York J. Med. (New York), 47: 2311-2315 (11 ref.), 1947.
 E – general – DC (add., infra-add., unspec. incr.) – post-mort. – drug-dep. humans – blood comp.,
 sites, lymph – G.I. tract – liver, kidney – anti-infectants – *CAAAL-0
 A-0746.

Detailed findings are presented of 7 cases of carbon tetrachloride poisoning. Special mention is made of the susceptibility of alcoholics, obese persons, undernourished individuals, and those ill with diabetes, liver, or renal disease. Both of the fatal cases concerned men who were alcoholics; for 1 of these persons, there was reason to believe that the continuous intake of alcohol, over a period of many months and years following the symptoms of carbon tetrachloride intoxication, was probably the cause of the progressive changes in the liver, long after all the acute manifestations of carbon tetrachloride poisoning had subsided. In the other fatal case, death resulted 10 days after carbon tetrachloride exposure.

508. Greenberg, L., and Ingalls, J. W.
 EFFECT OF DRUGS ON SURVIVAL TIME FROM SCORPION ENVENOMATION.
 J. Pharm. Sci. (Washington), 52(2): 159-161 (14 ref.), 1963.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in
 vivo – dose resp. – CNS – *CAAAL-11165-D2
 A-0747.

A lethal dose (0.2 ml) of diluted venom of the North African scorpion, *Androctonus australis*, was administered ip to mice. 30 min prior to the venom dose, 0.1 ml of a 20% alcohol sol was administered to some of the mice. The alcohol consistently reduced the survival time of the mice (14.9 ± 0.23 min, as compared to 20.6 ± 1.01 min for controls). The effects of 14 other drugs were also studied.

509. Greenberg, L. A., and Lester, D.
 ALCOHOL BREATH TESTS AND BREATH DEODORIZATION BY CHLOROPHYLL DERIVATIVES.
 Quart. J. Stud. Alcohol (New Haven), 15: 16-20 (2 ref.), 1954.
 E – exp. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – other drug lev. –
 *CAAAL-6817-U1
 A-0748.

4 adult male subjects received 5 oz of 100-proof whiskey on two separate occasions; on the second occasion 50 mg of sodium potassium copper chlorophyllin was given 75 min, and again 135 min, after the whiskey. Blood samples were taken 60, 90, 120, 150, and 180 min after drinking, and breath

alcohol was analysed simultaneously. A similar experiment was made after the drinking of 24 oz of beer. Chlorophyllin had no effect on blood alcohol levels, or on the blood alcohol values derived from breath analyses.

510. Greenberg, L. A., and Carpenter, J. A.
 THE EFFECT OF ALCOHOLIC BEVERAGES ON SKIN CONDUCTANCE AND
 EMOTIONAL TENSION. I. WINE, WHISKY AND ALCOHOL.
 Quart. J. Stud. Alcohol (New Haven), 18: 190-204 (11 ref.), 1957.
 E – exp. comp. – congen. stud. – humans – acute admin. – in vivo – blood lev. – nerv. syst. – skel.,
 muscle, skin – *CAAAL-8264-J1 A-1397.

Experiments were performed on 8 fasted male human subjects, to study the effects on emotional tension of 50 and 350 ml doses of red burgundy wine (12% alcohol by vol) and 12% ethanol sol, by measuring the basic skin conductance (BSC) under conditions of uniform and sustained activity, and the magnitude of the galvanic skin response (GSR) evoked by momentarily applied emotional stimuli given during otherwise uniform activity. It was found that 50 ml of wine had no significant effect on BSC, while 350 ml markedly lowered it. 50 ml of ethanol moderately lowered BSC, but 350 ml failed to have any greater lowering effect than the smaller dose. Neither wine nor ethanol sol in the 50 ml dose had much effect on GSR, reducing it by 8% and 5%, respectively, whereas the 350 ml dose had a marked effect, even reducing it by 53% and 49% respectively. It is concluded that alcoholic beverages, even in doses insufficient to cause intoxication, can reduce emotional tension. The difference between the effects of the 50 ml doses of wine and ethanol sol is attributed to the different rates of alcohol absorption. The difference in effects of the higher dose of the beverages, however, is attributed to the disagreeable taste and gastric action of ethanol sol.

511. Greenberg, L. A.
PSYCHOPHARMACOLOGIC EFFECTS OF CONGENERS.
 Twenty-eighth International Congress on Alcohol and Alcoholism, Washington, D.C., U.S.A.,
 September 15-20, 12 pp. (0 ref.), 1968.
 E – presentation – review – congen. stud. – humans – mammals – blood lev. – mot. perform. – psychol.
 perform. – CNS – metab. proc. – *CAAAL-0 B-0310.

The results of the proceedings of a symposium held at the Rutgers Center of Alcohol Studies in 1968 are reported. The recent investigations into the acute, chronic, and hangover effects of the congeneric substances in distilled beverages, with respect to experimental psychology, neurophysiology, biochemistry, metabolism, and behavioural science are discussed and reviewed.

512. Greenberg, L. A.
 THE APPEARANCE OF SOME CONGENERS OF ALCOHOLIC BEVERAGES AND
 THEIR METABOLITES IN BLOOD.
 Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 20-25 (15 ref.), 1970.
 E – exp. comp. – congen. stud. – mammals – acute admin. – in vivo – blood lev. – metab. proc. –
 alcohols – *CAAAL-12885-A2 B-0520.

Ethyl acetate and isoamyl alcohol, the 2 most abundant congeners in distilled spirits, and their metabolic products in blood were studied following ip administration in rats. 3 groups of 10 rats each were given the following solutions respectively: 4 g ethanol/kg body wt as a 20% ethanol sol (0.625 ml every 15 min for 4 doses) containing 600 mg isoamyl alcohol/l, the above ethanol sol containing 400 mg ethyl acetate/l, or water containing 600 mg isoamyl alcohol/l. Ethanol concentrations and relative amounts of isoamyl alcohol, isovaleraldehyde, and ethyl acetate in blood were then estimated every 2 hr by gas chromatographic analysis. Isoamyl alcohol, given alone, was rapidly metabolized, and was detectable only for 2 hr in blood; its intermediate metabolite, isovaleraldehyde, was never

detected. Following isoamyl alcohol-ethanol administration, blood isoamyl alcohol was detectable for 10 hr, and isovaleraldehyde for 8 hr, suggesting that low concentrations of the latter 2 unsuccessfully competed with ethanol and acetaldehyde for the same metabolic enzymes. Following ethyl acetate-ethanol administration, blood ethanol concentrations resembled those after isoamyl-ethanol administration, and ethyl acetate was not detected after 2 hr, suggesting different metabolic enzyme systems for ethanol and ethyl acetate. It is concluded that congeners and their metabolites possibly accumulate and interact metabolically and pharmacologically with ethanol during prolonged drinking to produce persisting undesirable effects.

513. Greenberg, R. E., Goldstein, L., and Pfeiffer, C. C.
 COUNTERACTION OF THE EFFECTS OF ACUTE ETHANOL INTOXICATION BY CNS
 STIMULANTS & TERTIARY AMINES.
 Pharmacologist (Detroit), 6(2): 170 (1 ref.), 1964.
 E – abst. – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – blood
 lev. – CNS – metab. proc. – amphetamines – hallucinogens – hormones, hormone antag. – stimulants
 – *CAAAL-0 A-0749.

The effects of certain stimulants and non-stimulant tertiary amines upon the CNS depression caused by acute ethanol intoxication were investigated in rabbits. LSD (5 mcg/kg), amphetamine (5 mg/kg, the tertiary amine of carnitine), and deanol (100 mg/kg) were administered just prior to ethanol infusion (ethanol dosage was calculated to give a blood level of 300 mg%). The data indicate that the acute cortical depression produced by ethanol may be significantly reversed by the action of stimulants. In the case of deanol, the reversal may occur by chemical combinations with ethanol to form inactive compounds. The above report is also discussed in: Anonymous (Medical News), J.A.M.A. (Chicago) 189(12): 36-37, 1964.

514. Greenberg, R. E.
 PREVENTION OF ALCOHOL-INDUCED CORTICAL DEPRESSION WITH
 STIMULANTS AND TERTIARY AMINES.
 Quart. J. Stud. Alcohol (New Haven), 28: 1-10 (15 ref.), 1967.
 E – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – absorp.,
 distrib., stor. – CNS – metab. proc. – nerv. syst. – amphetamines – hallucinogens – stimulants –
 *CAAAL-11223-D2 B-0311.

2 series of experiments with rabbits were conducted to determine: (1) the effect of alcohol on the quantitated cortical electroencephalogram, (2) the effects of stimulant drugs on the alcohol-produced changes and on blood alcohol levels, and (3) the possible modes of action of the stimulants. Alcohol infusions sufficient to produce a blood level of 300 mg% were administered with lysergide (5 µg/kg), d-amphetamine (6 mg/kg), desmethyl carnitine (100 mg/kg), or dimethylaminoethanol (100 mg/kg). All of the drugs prevented alcohol-induced cortical depression—lysergide prevented 98% of the depression; d-amphetamine, 97%; desmethyl carnitine, 87%; and dimethylaminoethanol, 80%.

515. Greenberg, R. S., and Goldstein, L.
 AN EEG STUDY OF THE RELATIONSHIPS BETWEEN BRAIN STRUCTURES IN
 RABBITS UNDER ETHANOL AND D-AMPHETAMINE.
 Quart. J. Stud. Alcohol (New Haven), 30: 843-848 (13 ref.), 1969.
 E – exp. cont. – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – psychol. perform.
 – CNS – amphetamines – *CAAAL-12776-D2 B-0521.

The effect of ethanol and d-amphetamine on the bioelectrical activity of the parietal cortex (C), midbrain reticular formation (RF), ventral hippocampus (H) and the relationships between these

structures, were studied by electroencephalograph (EEG) in rabbits. After implantation of the 3 EEG electrodes and drug administration were completed, the rabbits were set in a sound-attenuated box for a 30 min EEG recording. 24 trials were conducted, including 15 with ethanol (1 g or 2 g/kg of body weight in distilled water to a vol of 50 cc po), 6 with ethanol and amphetamine (2.5 mg/kg iv) in combination, and 2 with water (50 cc distilled, po). The data indicate that ethanol alone changes the normal relationship of some brain structures (RF-C and RF-H) without apparently affecting others (H-C), to produce the cortical activity and behavior characteristic of ethanol inebriation. D-amphetamine produces further changes in the relationship of structures (H-C), to generate an "arousal" effect on the EEG without demonstrably reversing the sedative effect of ethanol.

516. Greenfield, A. R.

A NEW TYPE OF SEDATION FOR THE ACUTE ALCOHOLIC.

American Practitioner and Digest of Treatment (Philadelphia), 7(2): 241-244 (2 ref.), 1956.
E – general – case hist. – DC (unchanged) – drug-dep. humans – in vivo – mot. perform. – psychol. perform. – cardiovasc. – CNS – tranquilizers – *CAAAL-7575-N11 A-1281.

55 acutely-intoxicated chronic alcoholics, characterized by anxiety, tension, or depression, were treated with reserpine; 27 were hospitalized, and 28 treated as outpatients. Initial parenteral injections of 2.5 mg reserpine on the first day had an onset within 2 hr, and lasted up to 24 hr; where necessary, this administration was repeated later on the same day and on days 2 and 3. 0.25 mg reserpine was then given po 3 times/day. Chloral hydrate or barbiturates were used temporarily in some difficult cases, and oxyphenonium bromide, an anticholinergic, helped to decrease gastric irritability and spasm, and to counter possible reserpine side-effects, such as nasal stuffiness. Treatment was successful in 51 of the 55 cases, the 4 failures being non-cooperative outpatients. Besides occasional nasal stuffiness, the only other side-effect was a mild to moderate hypotensive episode, which occurred in 6 patients on parenteral reserpine, and which was corrected by simple postural techniques. Reserpine appears to be very useful in treating the acute alcoholic phase, and has advantages over other sedatives by being non-addicting and non-incapacitating. No combined effect of alcohol and reserpine is noted.

517. Greenfield, A. R.

PERPHENAZINE IN THE MANAGEMENT OF ACUTE ALCOHOLIC INTOXICATION.

Curr. Ther. Res. (New York), 3(5): 217-220 (13 ref.), 1961.
E – exp. – conj. addict. – DC (unspec.) – psychot. humans – drug-dep. humans – cardiovasc. – CNS – G.I. tract – tranquilizers – *CAAAL-9413-N31 A-0750.

70 patients (24 women) in a state of acute alcohol intoxication received perphenazine. The first few doses were administered im, and 5-10 mg administered on the first day. The usual dose of the following days (never for more than a 5-day period) was 5 mg/day. Thereafter, the drug was given po, 16 mg daily. The calming effect of perphenazine became apparent within 20 to 30 min, and satisfactory control was obtained in all patients. It is concluded that perphenazine is a particularly effective drug for the symptomatic management and control of acute alcohol intoxication.

518. Greenhouse, H. R., and Pilot, M. L.

RESERPINE AS AN ADJUNCT IN THE TREATMENT OF ALCOHOLISM.

Quart. J. Stud. Alcohol (New Haven), 18: 468-474 (4 ref.), 1957.
E – SEC – exp. – case hist. – DC (add., infra-add., unspec. incr.) – psychot. humans – drug-dep. humans – psychol. perform. – tranquilizers – *CAAAL-8414-M1 A-0751.

During a 12-month period, reserpine was used at the New Haven Outpatient Clinic of the Connecticut Commission of Alcoholism, to help reduce the symptoms that develop following withdrawal of alcohol. In 32 of the 71 cases, reserpine seemed to help the patient achieve a measure of sobriety. 3 things were noted clinically: firstly, the patients who maintained sobriety with reserpine treatment

testified to the relief of anxiety; secondly, compared with other medications, reserpine seemed very effective during the early stages of treatment; and thirdly, reserpine seemed to potentiate the effect of alcohol in patients who continued to drink.

519. Greiser, E., and Soehring, K.

DIE AUFNAHME VON PENTOBARBITAL DURCH MENSCHLICHE ERYTHROCYTEN IN VITRO UND IHRE BEEINFLUSSUNG DURCH ÄTHANOL. [The effect of ethanol on the absorption of pentobarbital by human erythrocytes in vitro].

Naunyn Schmiedeberg. Arch. Pharm. Exp. Path. (Berlin), 255(1): 17 (0 ref.), 1966.
G – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vitro – absorp., distrib., stor. – blood comp., sites, lymph – barbiturates – *CAAAL-0 B-0312.

Ethanol (0.083 M/l = 4°/oo) was found to impede significantly, during the first 2 min of a 6-min-long incubation period, the absorption of pentobarbital by human erythrocytes in vitro. A change in the membranes may be involved. In addition to passive diffusion, it seems likely that pentobarbital is also absorbed through membranes.

520. Greiser, E.

KOMBINATIONSWIRKUNGEN VON ARZNEIMITTELN UND ALKOHOLO: EIN BERICHT ÜBER DIE ARBEITSTAGUNG DER SEKTION ARZNEIMITTEL UND VERKEHR DER DEUTSCHEN GESELLSCHAFT FÜR VERKEHRSMEDIZIN E. V. AM 26./27. MÄRZ 1966 IN BAD OEYNHAUSEN. [Interaction of drugs and alcohol: a report on the conference of the Drugs and Traffic Section of the German Association for Automotive Medicine, Inc., March 26-27, 1966, in Bad Oeynhausen].

Deutsche Apotheker-Zeitung (Stuttgart), 106(21): 752-754 (0 ref.), 1966.
G – review – DC (add., infra-add., unspec. incr.) – DC (sensit.) – med.-leg. – mot. vehic. – humans – mammals – blood lev. – cardiovasc. – CNS – metab. proc. – analg., antipyret. – anesthetics – barbiturates – stimulants – tranquilizers – *CAAAL-0 B-0313.

Papers presented to the conference, containing clinical, experimental, and statistical data on the effects of alcohol-drug combinations, are reviewed. The medico-legal problem, drug addiction complications, and the interaction of alcohol with barbiturates, tranquilizers, stimulants, opiates, and other compounds, are discussed. The essential difference between synergism and intolerance reactions is explained. It is pointed out that the danger of synergism, apart from driving impairment, is the accidental lethal intoxication. This conference is also reported by E. Greiser, in Zeitschrift für Praktische Anästhesie und Wiederbelebung (Stuttgart), 1(4): 265-269, 1966.

521. Greiser, E.

KOMBINATIONSWIRKUNGEN VON ARZNEIMITTELN UND ALKOHOLO: EINE ARBEITSTAGUNG DER SEKTION „ARZNEIMITTEL UND VERKEHR“ DER DEUTSCHEN GESELLSCHAFT FÜR VERKEHRSMEDIZIN E. V. AM 26. UND 27. MÄRZ 1966 IN BAD OEYNHAUSEN. [Interaction of drugs and alcohol: a conference of the Drugs and Traffic Section of the German Association for Automotive Medicine, Inc., March 26 and 27, 1966, in Bad Oeynhausen].

Zeitschrift für Praktische Anästhesie und Wiederbelebung (Stuttgart), 1(4): 265-269 (0 ref.), 1966.
G – review – DC (add., infra-add., unspec. incr.) – DC (sensit.) – med.-leg. – mot. vehic. – humans – mammals – blood lev. – cardiovasc. – CNS – metab. proc. – analg., antipyret. – anesthetics – barbiturates – stimulants – tranquilizers – *CAAAL-0 A-0314.

Papers presented to the conference, containing clinical, experimental, and statistical data on the effects of alcohol-drug combinations, are reviewed. The medico-legal problem, drug addiction complications,

and the interaction of alcohol with barbiturates, tranquilizers, stimulants, opiates, and other compounds, are discussed. The essential difference between synergism and intolerance reactions is explained. It is pointed out that the danger of synergism, apart from driving impairment, is the accidental lethal intoxication. This conference is also reported by E. Greiser in *Deutsche Apotheker-Zeitung* 106(21): 752-754, 1966.

522. Greiser, E., and Soehring, K.

DER TRANSPORT VON PENTOBARBITAL DURCH BIOLOGISCHE MEMBRANEN UND SEINE BEEINFLUSSUNG DURCH ÄTHANOL. [The transport of pentobarbital through biologic membranes, and the effect of ethanol on it].

Arzneimittelforschung (Aulendorf), 17: 207-214 (62 ref.),

1967.

G – ES – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – in vitro – dose resp. – absorp., distrib., stor. – blood comp., sites, lymph – liver, kidney – metab. proc. – barbiturates – *CAAAL-0

B-0315.

Tests on erythrocytes and renal slices of guinea pigs were made in order to study the changes in membrane permeability caused by ethanol. Pentobarbital was absorbed at $\pm 2^{\circ}\text{C}$. Higher temperatures caused a temperature- and energy-related increase of the permeation rate. This second transport was inhibited by sodium azide as well as by ethanol (0.083 M). The absorption of pentobarbital was inhibited at low substrate concentrations, whereas it was increased in the presence of high ones. It is suggested that ethanol may change the repartition of feeble acids between the cells and the intercellular spaces.

523. Grier, W. F.

THE TOXIC EFFECTS OF ANTIFEBRIN COMPLICATED WITH ALCOHOLISM, ILLUSTRATED BY A CASE.

Medical Record (New York), 42(18): 511 (0 ref.),

1892.

E – general – case hist. – DC (antidotal) – DC (add., infra-add., unspec. incr.) – drug-dep. humans – cardiovasc. – G.I. tract – respir. – elect., water-bal. agents – analg., antipyret. – cardiovasc. agents – sed., hypnot. – *CAAAL-0

A-0752.

A case history is given of a female dyspso-maniac who indulged in a variety of alcoholic beverages and medicinal remedies, and had taken 40 grains of antifebrin (acetanilide) within 11 hr; when treatment was begun, the patient was intoxicated and cyanosed. The case was treated as one of acute alcoholism—initially, 10 grains calomel, followed by 1% nitroglycerine sol (1/2 drop/hr), and, on the second day, 10 minim doses of tincture of digitalis, 20 grains chloral, 30 grains ammonium bromide, and a saline purgative. By the third day, the cyanosis had disappeared, and, by the fifth day, the patient's health was normal.

524. Grilichess, R.

ÜBER DIE PHARMAKOLOGISCHE WIRKUNG KOMBINierter URETHANE UND ALKOHOLE. [The pharmacologic effect of combined urethanes and alcohols].

Zeitschrift für Allgemeine Physiologie (Jena), 15: 468-478 (12 ref.),

1913.

G – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – neoplast. agents – *CAAAL-0

A-0753.

Bürgi's Law (which states that 2 drugs, belonging to the same chemical group, can have a potentiating effect only if they belong to different subgroups which act in different ways on the mammalian body; if they act in the same way, the effect is only additive) was investigated in rabbits. In the first experiment, the effect of simultaneous sc injection of ethyl and methyl urethane (various doses) was found to be additive. In the second experiment, ethyl urethane (0.5 g/kg) and methyl urethane (1-4 g/kg), alone and in different combinations with ethanol (3-8 cc 10% sol/kg) and methanol (4-10 cc

10% sol/kg), were administered sc and im. Only additive effects were observed, and Bürgi's Law was confirmed.

525. Groves, J. W.

POISONING BY MORELS WHEN TAKEN WITH ALCOHOL.

Mycologia (New York), 56: 779-780 (0 ref.),

1964.

E – general – case hist. – DC (sensit.) – humans – cardiovasc. – *CAAAL-0

A-1282.

Species of the genus *Coprinus* have been most often implicated in reactions due to alcohol-fungi interaction, but other fungi may also have this effect. A case is described in which a man collected a pound of narrow-capped morels, eliminating the poor ones, and prepared them the next day by soaking them in salt water. They were eaten fried at the noon meal by the man, his wife and 2 daughters, all of whom had equal portions. That afternoon, the man and his wife each drank about 2 oz of rye, while the daughters had soft drinks. Within 20 min, the wife became violently ill with diarrhoea, severe emesis, and vomiting; her pulse rate was 120 at the height of the attack. The husband followed 5 min later with similar symptoms. Both had 3 or 4 violent episodes, then felt better and recovered within several hr. The daughters, being unaffected, provided a good control. It seems improbable that other fungi were inadvertently consumed. No agarics were picked, and the symptoms were not those of *Gyromitra* poisoning.

526. Gruber, C. M., Jr.

A THEORETICAL CONSIDERATION OF ADDITIVE AND POTENTIATED EFFECTS BETWEEN DRUGS WITH A PRACTICAL EXAMPLE USING ALCOHOL AND BARBITURATES.

Arch. Int. Pharmacodyn. (Gand), 102(1-2): 17-32 (11 ref.),

1955.

E – exp. cont. – review – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – species or sex diff. – absorp., distrib., stor. – CNS – liver, kidney – metab. proc. – respir. – barbiturates – *CAAAL-7277-D2

A-0754.

The literature on ethanol-drug interaction is reviewed. Certain difficulties in the interpretation of responses and effects are discussed from the standpoint of the problems which arise in determining whether drugs with similar actions are synergistic or additive. The sleeping times and deaths (within 12 hr after drug) of white rats were recorded following ip doses of 95% ethanol (0.0, 1.95, 2.34, or 2.81 mg/kg), injected 1/2 hr prior to ip secobarbital sodium (0.0, 50, 60, 72, 86.7, 104, 125, or 150 mg/kg). Sleeping times were also determined after 95% ethanol (1.5, 1.8, 2.1, 2.5, or 3.0 cc/kg), given in a single ip injection with sodium phenobarbital (46.25, 55.5, 66.7, 80.0, or 96 mg/kg, respectively), and the deaths recorded after combined administration in which the ratios of pentobarbital (66.7-168 mg/kg) to alcohol (2.1-5.1 cc/kg) were 4:0, 3:1, 1:3, and 0:4. In all combinations, there was no evidence of more than an additive effect.

527. Grugni, C.

L'ANESTESIA NELL'ALCOOLIZZATO. [Anesthesia in the alcoholic].

Minerva Anest. (Turin), 34: 1368-1375 (8 ref.),

1968.

I – ES – general – DC (add., infra-add., unspec. incr.) – DC (unspec.) – cardiovasc. – CNS – G.I. tract – glands – liver, kidney – skel., muscle, skin – analg., antipyret. – anesthetics – barbiturates – cardiovasc. agents – sed., hypnot. – tranquilizers – *CAAAL-0

B-0936.

Because of the delicate state of alcoholics, who may be chronically undernourished, suffer from polyvitaminic deficiencies, and have parenchymal damage, anesthesia presents serious complications. In intoxicated cases, 100-200 mg chlorpromazine is recommended to decrease excitability. For induction of narcosis, pentothal, followed by oxygen and nitrous oxide, is the most suitable. To maintain narcosis, fluothane, in concentrations of less than 1%, with meperidine is suggested. Other

halogenic anesthetics, such as chloroform, are rejected. In the case of severe liver damage, local anesthesia with dehydrobenzperidol and an analgesic with selective action, such as fentanyl, is recommended: alternatively, γ -aminobutyric acid plus an analgesic can be used. Delirium tremens is the most difficult complication, and has a mortality rate of about 20%. The iv administration of 5-10% glucose sol, B vitamins, sodium, potassium, and cortisone is advised, in addition to sedation with pentothal or meprobamate.

528. Grüner, O., and Federlin, C.

ÜBER DEN VERLAUF DER BLUTALKOHOLKURVE NACH COFFEINGABEN. [On the course of the blood alcohol curve after caffeine administration].

Blutalkohol (Hamburg), 3:188-199 (32 ref.),

1965.

G – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev.

– metab. proc. – stimulants – *CAAAL-0

B-0316.

10 human subjects divided into 4 groups received 1 g ethanol/kg po. Groups A and B received 250 mg caffeine sodium benzoate sc or drank 1 cup of strong coffee, 1 group receiving the caffeine 28 min, and the other group 185 min after the alcohol. Groups C and D served as controls and received 1 cup of caffeine-free coffee 28 or 185 min after the alcohol. Blood alcohol determinations were made at various intervals. In A and B, the blood alcohol curve rose more slowly in the absorption phase, and fell more slowly in the elimination phase. In C and D the curves were normal. Possible reasons for the changes in A and B are discussed.

529. Grüner, O., Ludwig, O., and Bauch, G.

ÜBER DEN VERLAUF DER BLUTALKOHOLKURVE NACH NIKOTINGENUSS. [On the course of the blood alcohol curve after nicotine intake].

Blutalkohol (Hamburg), 5(4): 254-263 (26 ref.),

1968.

G – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev.

– *CAAAL-0

B-0317.

Tests were carried out to determine the influence of smoking on the blood alcohol curve in 11 subjects given 1 g/kg absolute alcohol as a 20% sol. The subjects were made to fast 8-10 hr prior to test and abstain from alcohol 18 hr prior to test. Blood alcohol curves were determined according to the Widmark method. The results showed that smoking slowed down the ascent of the absorption curve, with a clear decrease in the maximum peak, i.e., the curve was flatter than that of the control. The results are interpreted as an indication of a nicotine-conditioned absorption slow-down.

530. Grymiński, J., Lyczewska, J., Styszewska, H., and Walczak, J.

OCENA HEPATOTOKSYCZNEGO DZIAŁANIA LEKÓW PRZECIWPRAŁKOWYCH U CHORYCH NA GRUŻLICĘ PŁUC NADUŻYWAJĄCYCH ALKOHOLU. [Assessment of the hepatotoxicity of antituberculous drugs in tuberculous patients who are excessive drinkers].

Gruźlica i Choroby Płuc (Warsaw), 37(8): 749-757 (10 ref.),

1969.

Po – ES – RS – exp. comp. – general – case hist. – humans – drug-dep. humans – chronic admin.

– in vivo – blood lev. – other drug lev. – liver, kidney – anti-infectants – *CAAAL-14430

B-1012.

The hepatotoxic effects of various tuberculostatic agents were assessed in clinical examinations and liver function tests made on 55 excessive-drinking, tuberculous patients. Liver function was normal in 39 patients (group 1), and impaired in 16 subjects (group 2). Over a 2- to 12-month period of chemotherapy with streptomycin (SM), isoniazid (INH), para-aminosalicylic acid (PAS), ethionamide (ETA), pyrazinamide (PZA), viomycin (VM), and cycloserine (CS), 10 patients developed hepatic disturbances, 5 of them showing severe and protracted liver damage. Most (8 out of 10 subjects) hepatic disturbances occurred in patients who originally exhibited no liver abnormalities,

probably due to the fact that more subjects received PZA in group 1 (13) than in group 2 (3). Hepatotoxicity occurred predominantly with PZA (5 cases); the causative agent was PAS in 2 cases, ETA in 1 case, and PZA, ETA, or PAS in 2 cases. No hepatotoxic effect was found with INH, SM, VM, or CS. Attention is called to the considerable incidence of hepatic disturbances resulting from PZA administration to excessive drinkers. ETA is contraindicated in cases of severe liver damage, but not if the damage is only slight.

531. Gugler, R.

ALKOHOL UND MEDIKAMENTE, ARZT UND PATIENT. [Alcohol and drugs, the doctor and the patient].

Med. Welt (Stuttgart), 33: 1764-1768 (20 ref.),

1968.

G – general – stat. surv. – case hist. – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev. – mot. perform. – CNS – antidepressants – barbiturates – tranquilizers – *CAAAL-0 B-0522.

A general review of the problem of interaction between alcohol and commonly prescribed drugs and patent medicines (primarily concerning effects on driving ability) is given for physicians. Narcotics, sleeping pills, tranquilizers, psychomedicines, antiepileptics, antihistamines, stimulants, diet pills, and drugs to reduce blood pressure and spinal muscle tension are considered. Statistics on 3,782 blood samples, taken in 1965, 1966, and the first part of 1967 showed that 416 people had taken both alcohol and drugs within 24 hr of sampling. Of 442 who took drugs, 29 were found to have markedly decreased efficiency as a result. 44% of the 442 had taken analgesics, often in very high doses, many of which were brand name compounds containing barbiturates. A table is included showing the increased effects of 200 mg/kg alcohol in the presence of novril, imipramine, chlorpromazine, phenobarbital and librium. Case histories of 5 traffic offenders who experienced alcohol-drug interactions are included.

532. Guild, W. R., Young, J. V., and Merrill, J. P.

ANURIA DUE TO CARBON TETRACHLORIDE INTOXICATION.

Ann. Intern. Med. (Philadelphia), 48(6): 1221-1227 (9 ref.),

1958.

E – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – other drug lev. – absorp., distrib., stor. – acid-base, blood pH, elect. – blood comp., sites, lymph – cardiovasc. – G.I. tract – liver, kidney – metab. proc. – respir. – anti-infectants – *CAAAL-0 A-0755.

10 cases (5 fatal) of acute CCl_4 intoxication—16 by inhalation and 4 by ingestion—resulting in anuria are reported. 1 of the 20 patients had consumed large quantities of alcohol daily for a period of months or years; of these, 14 admitted taking alcohol before, during, or after CCl_4 exposure, and 2 light drinkers had also taken alcohol at the time of exposure. In several cases, fellow workers were also exposed but suffered no ill effects; in at least 1 case, the fellow worker had not consumed alcohol. The author considers that ingestion of alcohol and CCl_4 simultaneously may increase CCl_4 absorption; this may also be the case if exposure is by inhalation, and if alcohol is being excreted by the lungs or is in significant concentration in the blood. Alcohol ingestion after CCl_4 exposure may not enhance CCl_4 absorption, but may possibly condense with phosgene oxidized from CCl_4 to form ethylchloroformate.

533. Gumbel, B.

LÄSST SICH EIN ERHÖHTER ALKOHOLGEHALT DES BLUTES MEDIKAMENTÖS SENKEN? UNTERSUCHUNGEN UNTER BESONDERER BERÜCKSICHTIGUNG DES MITTELS „CONTRA“. [Can the alcohol level in the blood be lowered by means of a drug? Studies with particular consideration of the drug "Contra"].

Deutsch. Med. Wschr. (Stuttgart), 81(46): 1850-1853 (23 ref.),

1956.

G – exp. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – metab. proc. – nerv. syst. – *CAAAL-8072-A1 A-0756.

The sobering effect of the new anti-alcohol drug contra was tested. 10 human subjects drank enough wine to achieve a blood alcohol level of 1-2°/oo, and ingested contra 10-15 min after the desired blood level was reached. Blood samples were taken every 15-30 min and analyzed for alcohol by the Widmark and alcohol dehydrogenase methods. The oxidation rate was calculated to range between 0.11 and 0.24°/oo/hr. Since this is within the normal range, it is concluded that contra did not accelerate alcohol elimination. Clinical signs, such as rotation nystagmus, were not affected by the drug, and reaction times were not improved. Subjective observations did not show any sobering effects of the drug. It is concluded that contra cannot lower blood alcohol levels or improve impaired performance.

534. Gupta, R. C., and Kofoed, J.

TOXICOLOGICAL STATISTICS FOR BARBITURATES, OTHER SEDATIVES, AND TRANQUILLIZERS IN ONTARIO: A 10-YEAR SURVEY.

Canad. Med. Ass. J. (Toronto), 94: 863-865 (3 ref.),

1966.

E – SEC – stat. surv. – DC (add., infra-add., unspec. incr.) – humans – blood lev. – other drug lev. – barbiturates – *CAAAL-0 B-0148.

A survey of Ontario by the Attorney-General's Laboratory, Toronto, for the period 1955-1964, showed a large increase in the number of cases of poisoning due to barbiturates, tranquilizers, and non-barbiturate sedatives. Urine and blood samples from persons charged with driving motor vehicles while under the influence of drugs were also examined. Doses of barbiturates and alcohol much smaller than the lethal doses can cause death when taken in combination. Statistics revealed that as little as 0.5 mg% of secobarbital or pentobarbital and 1.0 parts/1000 of alcohol in the blood can be fatal. Of 143 deaths in 1964 from barbiturate poisoning, 50 showed the presence of alcohol in postmortem examination.

535. Gus'kov, V. S.

OSOBENNOSTI OKISLENIIA ALKOGOLIA PRI SHIZOFRENII. [Characteristics of alcohol oxidation in schizophrenia].

Gosundarstvennyi Nauchno-issledovatel'skii Institut Psikhiatrii Ministerstva Zdravookhraneniia R.S.F.S.R., Trudy (Moscow), 27: 138-146 (0 ref.),

1961.

R – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – psychot. humans – drug-dep. humans – acute admin. – in vivo – blood lev. – metab. proc. – amphetamines – barbiturates – hormones, hormone antag. – tranquilizers – *CAAAL-10768-A1 A-0757.

Alcohol (0.5 g/kg, 10-40% sol) was given to 80 schizophrenics, 11 normal subjects, and 17 alcoholics. Blood alcohol levels were determined 20 min after alcohol and at hourly intervals thereafter for 7 hr. In the large majority of schizophrenics, reducing substances were initially higher than in normal or alcoholic subjects, and the time necessary to reach maximum alcohol concentrations, and for complete alcohol oxidation, were longer. Such deviation, seen particularly in hebephrenics and catatonics, was more pronounced at the onset or during acute periods, and almost disappeared during the chronic phase of the condition. Active therapy by injections of insulin (up to 30 units) or chlorpromazine (10-15 g) normalized the alcohol oxidation process in the blood, and decreased the level of reducing substances. Pervitin (0.01 g) was found to increase alcohol oxidation, and hexenal (1 g, im) to decrease it.

536. Gutschmidt, J.

GIBT ES ANTIRAUSCHMITTEL? [Are there any antidotes for alcoholic intoxication?].

Med. Klin. (Munich), 34(38): 1263-1264 (0 ref.),

1938.

G – general – DC (decrease) – DC (unchanged) – humans – blood lev. – cardiovasc. – CNS – metab. proc. – *CAAAL-576-A7 A-0758.

In a general discussion on the topic, the author remarks that there appears to be no substance which can reduce the alcohol concentration in the blood. Ingestion of fruit can increase the reducing power of the blood, but a considerable amount of fruit would be necessary to offset the effect of 1 glass of beer. Strong coffee can be helpful, if intoxication is slight, by increasing the blood flow, thus bringing more oxygen to the brain and causing increased alcohol excretion through the lungs. Nevertheless, at a higher degree of intoxication, the increased blood flow also brings more alcohol to the brain and negates the effect of an increased oxygen supply. It is probably correct to conclude that some antidotes may influence the subjective feeling of the intoxicated person, but none have an objective effect on the blood alcohol level.

537. Haag, H. B., Finnegan, J. K., Larson, P. S., and Smith, R. B., Jr.
STUDIES ON THE ACUTE TOXICITY AND IRRITATING PROPERTIES OF THE CONGENERS IN WHISKY.
 Toxic. Appl. Pharmacol. (New York), 1: 618-627 (15 ref.), 1959.
 E – exp. cont. – exp. comp. – congen. stud. – mammals – acute admin. – in vivo – dose resp. – senses
 – indust. intox. – *CAAAL-9462-V2 A-0759.

The acute oral toxicities of a series of congeneric fractions prepared from whiskey, of the synthetic prototypes of the fractions, of furfural, and of purified ethyl alcohol were determined in rats. Of these materials, furfural was found to be the most toxic ($LD_{50} = 0.135 \pm 0.015$ g/kg), and pure ethyl alcohol the least toxic ($LD_{50} = 10.4 \pm 0.75$ g/kg). With the exception of the acid fraction, the effects of the other congeners occurred more rapidly, and, in surviving animals, disappeared more quickly than was the case with ethyl alcohol.

538. Hadji-Dimo, A. A., Ekberg, R., and Ingvar, D. H.
EFFECTS OF ETHANOL ON EEG AND CORTICAL BLOOD FLOW IN THE CAT.
 Quart. J. Stud. Alcohol (New Haven), 29(4A): 828-838 (23 ref.), 1968.
 E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – cardiovasc. – CNS
 – anesthetics – autonomic agents – barbiturates – *CAAAL-11647-D2 B-0318.

The effects of up to 20 ml of a 25% ethanol sol in iv doses of 0.15-0.25 g/kg every 20 min on regional cerebral blood flow (rcbf) and electroencephalograms (EEG) were investigated in immobilized, tracheotomized cats. In 5 cats anesthetized with nitrous oxide in 20% oxygen, the max increase of both the rcbf and the EEG frequency index occurred 30 min after ethanol administration. With continuing ethanol injections, a decrease of rcbf and EEG frequency index occurred, often with a sharp fall in arterial blood pressure. In 10 cats anesthetized with pentobarbital (40-60 mg/kg), none of the initial phenomena (increase of rcbf and the EEG frequency index) were observed. Larger doses of alcohol (2-3 g/kg), however, still provoked the late increase of slow waves in the EEG and a decrease of critical blood flow, together with a fall in arterial blood pressure.

539. Haffner, F.
ZUR PHARMAKOLOGIE UND PRAXIS DER STIMULANTIEN. [The pharmacology and use of stimulants].
 Klin. Wschr. (Berlin), 17(38): 1310-1311 (4 ref.), 1938.
 G – SEC – exp. comp. – general – DC (decrease) – mammals – acute admin. – in vivo – dose resp.
 – amphetamines – autonomic agents – stimulants – *CAAAL-0 A-0760.

A general discussion is given concerning stimulants. Experimental research on mice established that 0.005 g/kg amphetamine cancelled the effect of 0.1 g/kg chloral hydrate or of 1-2 g/kg alcohol; twice this dosage was needed for caffeine to achieve the same effect, and the caffeine effect lasted only half as long (2 hr as compared to 4 hr for amphetamine). Four times the above amphetamine dose was needed for ephedrine, veritol, suprifen, and picrotoxin to have an antagonistic effect on alcohol.

540. Haggard, H. W., Greenberg, L. A., Rakieten, N., and Cohen, L. H.
STUDIES ON THE ABSORPTION, DISTRIBUTION AND ELIMINATION OF ALCOHOL.
VII. THE INFLUENCE OF INHALATION OF OXYGEN AND CARBON DIOXIDE AND
CERTAIN DRUGS ON THE CONCENTRATION OF ALCOHOL IN THE BLOOD
CAUSING RESPIRATORY FAILURE.

J. Pharmacol. Exp. Ther. (Baltimore), 69(3): 266-271 (33 ref.), 1940.
E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute
admin. – in vivo – blood lev. – CNS – metab. proc. – respir. – amphetamines – analg., antipyret. –
barbiturates – sed., hypnot. – *CAAAL-2671-D2 A-0761.

The concentration of alcohol in the blood causing respiratory failure in rats was determined under the following 3 conditions: 1) during oxygen inhalation; 2) during dilute carbon dioxide inhalation; and 3) after administration of the following substances: caffeine (5-15 mg/kg, given before initial alcohol injection or approximately 1 hr before death), 2, 4-dinitrophenol (10 mg/kg, given 1 hr before death under repeated alcohol injections), strychnine (0.1-1.0 mg/kg, given before initial alcohol injection), amphetamine (3.0 mg/kg, given 1 hr before death under repeated alcohol injection), sodium bromide (100-1000 mg/kg, given 2-4 hr before initial alcohol injection), amytal (5-50 mg/kg, given 1 hr before death), morphine (5, 10, and 20 mg/kg), acetanilid (10 and 50 mg/kg, given 30 min before alcohol), phenacetin (16 mg/kg, 30 min before alcohol), antipyrine (16 mg/kg, 30 min before alcohol), and aspirin (20 and 60 mg/kg, 30 min before alcohol). All the drugs except morphine failed to affect the concentration of alcohol in the blood which caused respiratory failure. Morphine had a marked effect; the 5 mg/kg dose reduced the alcohol concentration in the blood at death from the average level of 9.30 mg/cc to 8.12 mg/cc, and the 10 and 20 mg/kg doses were followed by death at concentrations of 7.74 and 7.39 mg of alcohol/cc.

541. Haggard, H. W., Greenberg, L. A., and Carroll, R. P.
STUDIES IN THE ABSORPTION, DISTRIBUTION AND ELIMINATION OF ALCOHOL.
VIII. THE DIURESIS FROM ALCOHOL AND ITS INFLUENCE ON THE
ELIMINATION OF ALCOHOL IN THE URINE.

J. Pharmacol. Exp. Ther. (Baltimore), 71: 349-357 (25 ref.), 1941.
E – SEC – exp. cont. – congen. stud. – humans – acute admin. – in vivo – dose resp. – blood lev.
– other drug lev. – liver, kidney – *CAAAL-3322-A1 A-1398.

The elimination of alcohol in relation to amount ingested, alcohol diuresis, influence of diuresis on alcohol elimination, and concentration of alcohol in blood in relation to diuresis, were studied in male humans. The comparative diuresis caused by alcohol, gin, whiskey, port, and sherry was determined in 4 subjects. For each beverage, total alcohol was 24 g, and the vol as needed was brought to 180 cc with water. The average diuresis was 121 cc. None of the beverages gave a greater diuresis than that expected from the alcohol content, and the wines gave less. The effect of beer was studied in 2 subjects. 720 cc of beer containing 24 g alcohol were given, and the rate of secretion of urine was compared to that after the same amount of water. In 1 subject, the amounts of urine secreted 5 hr after beer were 689 and 721 cc, respectively, in 2 experiments, and, 5 hr after water, were 560 and 579 cc; the corresponding values for the second subject were 853 and 951 cc after beer, and 692 and 765 cc after water. It is concluded that, aside from its bulk of water, beer causes no greater diuresis than would be expected from the content of alcohol.

542. Haggard, H. W., Greenberg, L. A., and Cohen, L. H.
THE INFLUENCE OF THE CONGENERS OF DISTILLED SPIRITS UPON THE
PHYSIOLOGICAL ACTION OF ALCOHOL.

Quart. J. Stud. Alcohol (New Haven), 4(1): 3-56 (41 ref.), 1943.
E – exp. comp. – congen. stud. – humans – mammals – acute admin. – in vivo – blood lev. – absorp.,
distrib., stor. – CNS – G.I. tract – liver, kidney – metab. proc. – anti-infectants – elect., water-bal.

agents – gastrointest. agents – neoplast. agents – nutritive agents – stimulants – *CAAAL-3600-A1
A-0762.

Investigation of the effects of congeners on alcohol action in rats showed that the congeners do not add directly to the alcohol toxicity. The amount of spirit alcohol causing respiratory failure varied widely with the different distilled spirits (7.8-11.8 mg alcohol/g wt). The rate of alcohol metabolism and degree of intoxication varied according to the amounts of congeners present. An attempt was made to alter the toxicity (by altering the alcohol oxidation) of distilled spirits and pure ethanol in rats by administering the following substances (100 rats/substance): oxygen, carbon dioxide, 2, 4-dinitrophenol (0.1 mg ip), methylene blue (5 mg sc), methionine (10 mg ip), alloxan (10 mg ip), glycine (10 mg ip), cysteine (3 or 6 mg ip), ascorbic acid (10 mg ip), and glutathione (3 or 6 mg ip). The only 2 substances to cause appreciable alteration of the alcohol toxicity were cysteine and, in particular, glutathione.

543. Hald, J., Jacobsen, E., and Larsen, V.

THE ANTAGONISM BETWEEN ALCOHOL AND ACETALDEHYDE IN RABBITS.

Acta Pharmacol. (Copenhagen), 8(2): 164-170 (0 ref.),

1952.

E – exp. cont. – DC (decrease) – DC (sensit.) – mammals – acute admin. – in vivo – blood lev. – respir. – miscellaneous – neoplast. agents – stimulants – *CAAAL-6187-B2
A-0763.

To demonstrate a possible antagonism between alcohol and acetaldehyde in animals, rabbits weighing 2.5-3.5 kg were anesthetized with 1.5 g urethane/kg sc. Some animals were then given 1.5 g 20% alcohol/kg by stomach tube, resulting in an average blood alcohol concentration of 150 mg%. 2% acetaldehyde, at the rate of 2.0-4.0 mg/min, was infused into the blood stream; blood samples were taken, and respiration was measured. It was found that the antagonism between alcohol and acetaldehyde on the respiration was fairly pronounced—the effect of 1 mg% acetaldehyde was 4 times as great in animals without an elevated blood alcohol level as in animals with 100-150 mg% alcohol in their blood. A similar antagonizing effect of alcohol was not seen when the respiration was stimulated by carbon dioxide.

544. Hald, J., Jacobsen, E., and Larsen, V.

THE ANTABUSE-EFFECT OF SOME COMPOUND[S] RELATED TO ANTABUSE AND CYANAMIDE.

Acta Pharmacol. (Copenhagen), 8: 329-337 (4 ref.),

1952.

E – exp. cont. – DC (sensit.) – mammals – acute admin. – in vivo – blood lev. – mot. perform. – cardiovasc. – unclass. ther. agents – *CAAAL-6396-B2
A-1283.

The sensitization of rabbits to ethanol by doses of 13 drugs related to antabuse and cyanamide was tested. Thiuramdisulfide, tetramethylthiuramdisulfide, tetraethylthiuramdisulfide, tetrapropylthiuramdisulfide, tetrabutylthiuramdisulfide, potassium dimethyl dithiocarbamate, sodium diethyl dithiocarbamate, tetramethylthiuram monosulfide, tetraethylthiuram monosulfide, dioxanthogen, cyanamide, diethylcyanamide, and dicyandiamide were administered as aqueous sol or fine suspension via a stomach tube. Alcohol was administered by the same method, 1.5 g/kg in a 20% sol. Blood samples were taken at predetermined intervals, in order to test alcohol and acetaldehyde levels. The related compounds fell into 4 groups. Among the thiuramdisulfides, the primary amine had no effect. The effect decreased through the series tetramethyl-, tetraethyl-, tetrapropyl- and tetrabutylamine. Dithiocarbamates had a shorter effective span than their corresponding disulfides. In the third group, thiurammonosulfides, tetramethyl- and tetraethyl- had marked and prolonged effects. Cyanamide and its derivatives, the fourth group, gave varied results; cyanamide itself had a slight effect, and diethylcyanamide, being too toxic to test sufficiently, gave uncertain results. Dicyandiamide had no effect.

545. Hall, A. J.
 CAFFEINE IN THE TREATMENT OF ALCOHOLIC TOXEMIA.
 Medical News (Philadelphia), 83: 831-833 (10 ref.), 1903.
 E – general – case hist. – conj. addict. – DC (antidotal) – drug-dep. humans – cardiovasc. – CNS
 – G.I. tract – liver, kidney – metab. proc. – stimulants – *CAAAL-0 A-0764.

The author advocates the use of caffeine as a safe therapeutic agent that can produce definite results in treating alcoholic toxemia, citing 4 cases to illustrate his point. To further support his argument, the symptoms and pathology of alcoholic toxemia and the therapeutic effect and physiological action of caffeine are tabulated. The recommended caffeine dosage is 1 or 2 grains every 1, 2, or 3 hr for 24-48 hr. Although the author does not advocate caffeine as a specific in all cases of alcoholic toxemia to the exclusion of all other remedies, he considers caffeine to have a physiological antagonism to alcohol "as clearly marked and as easily demonstrated as that between belladonna and opium." If this is admitted, the contention that alcoholism is a disease must be abandoned, and the fact that it is a toxemia be conceded.

546. Haller, E.
 ÜBER VERSCHIEBUNG DER REAKTION DURCH ALKOHOL. [The shifting of the alcohol reaction].
 Dissertation, Medical Faculty of the University of Tübingen, West Germany, 68 pp. (0 ref.), 1949.
 G – SEC – exp. – absorp., distrib., stor. – anesthetics – miscellaneous – *CAAAL-0 A-0765.

Tests were made to determine the influence of alcohol on acid-base colour reaction. It was found that 75% alcohol exercises an appreciable influence on the colour reaction of strong acid indicators, and the base reactions were reversed by alcohol. Alcohol showed little influence on barbituric acids and phenols. Experiments with yeast were made to investigate the alcohol effects in cells. It was hypothesized that, in cellular systems, effects of alcohol could be observed with low alcohol concentrations, because of preferential distribution of alcohol in the cells, and because of absorption or solubility in lipoids. This hypothesis could not be proven. The author speculates that alcohol may change membrane permeability almost immediately, and that, therefore, other effects are concealed, and cannot be established with present methods. More research is necessary to establish relevant conclusions.

547. Halliwell, G., Quinton, R. M., and Williams, F. E.
 A COMPARISON OF IMIPRAMINE, CHLORPROMAZINE AND RELATED DRUGS IN VARIOUS TESTS INVOLVING AUTONOMIC FUNCTIONS AND ANTAGONISM OF RESERPINE.
 Brit. J. Pharmacol. (London), 23: 330-350 (52 ref.), 1964.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – acute admin.
 – in vivo – CNS – metab. proc. – antidepressants – tranquilizers – *CAAAL-0 A-0766.

7 structurally-related compounds (imipramine, desmethylinipramine, amitriptyline, promazine, desmethylpromazine, chlorpromazine, and chlorprothixene) were examined in a series of tests involving autonomic functions and antagonism of reserpine. In 1 of the tests, albino male mice (16-22 g) were given 1 of the drugs (20 mice/drug dose) po 30 min before administration of 2.5 ml/kg ethanol in 20% sol po; all drugs except desmethylinipramine prolonged the sleeping-time when given in adequate dosage, but their potencies varied markedly. Sleep for 6 and 9 min was induced in only 2 of 55 controls receiving ethanol alone. Chlorpromazine and chlorprothixene were very active, inducing, in combinations with ethanol, sleep of 60 min duration in doses of about 1 mg/kg. Promazine and desmethylpromazine were slightly less potent (4-6 mg/kg), with the activity of the latter increasing less at higher doses than of the former. Amitriptyline (12 mg/kg) showed weak activity. Imipramine and, in particular, desmethylinipramine were the least potent drugs in the test, doses of 50 mg/kg inducing sleep with durations of only 49.4 ± 9.5 and 7.1 ± 3.7 min respectively.

548. Hameau

QUELQUES EXPÉRIENCES À PROPOS DE L'EMPLOI DE L'ALCOOL COMME ANTIDOTE DE LA STRYCHNINE. [Some experiences concerning the use of alcohol as an antidote of strychnine].

Gazette Médicale de Bordeaux (Bordeaux), 7: 330-332 (0 ref.),

1878.

F – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – CNS – stimulants – *CAAAL-0 A-0767.

A 3 month-old rabbit was injected with strychnine sulphate sol (0.01 g in 1 g of distilled water) in the auricle of the ear. 20 min later, the animal went into convulsions, then shock. 5 min after apparent death, the rabbit was given 1 g of 90% alcohol in the other ear. In less than 3 min, the animal's limbs relaxed and drew into convulsive flicks. In 25 min, the animal could support itself on its legs, had no convulsions, spontaneous or provoked, and began eating. The following day, it was found in perfect health. Another rabbit of the same age was injected in the same way with one g of 90% alcohol. 1 hr later, it went into a stupor and refused to eat. 25 hr later, it ate a little, then died in the night. The author feels that the efficacy of alcohol injections in strychnine poisoning is demonstrated by these experiences, and feels that alcohol may be considered as an antidote to the poison, or as a powerful sedative, the action of which on the CNS is antagonistic to strychnine.

549. Hameau

QUELQUES EXPÉRIENCES À PROPOS DE L'EMPLOI DE L'ALCOOL COMME ANTIDOTE DE LA STRYCHNINE. [Some experiences concerning the use of alcohol as an antidote of strychnine].

Société de Médecine et de Chirurgie de Bordeaux, Mémoires et Bulletins (Bordeaux), 372-375 (0 ref.),

1878.

F – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – CNS – stimulants – *CAAAL-0 A-0768.

A 3 month-old rabbit was injected with strychnine sulphate sol (0.01 g in 1 g of distilled water) in the auricle of the ear. 10 min later, the animal went into convulsions, then shock. 5 min after apparent death, the rabbit was given 1 g of 90% alcohol in the other ear. In less than 3 min, the animal's limbs relaxed and drew into convulsive flicks. In 25 min, the animal could support itself on its legs, had no convulsions, spontaneous or provoked, and began eating. The following day, it was found in perfect health. Another rabbit of the same age was injected in the same way with one g 90% alcohol. 1 hr later, it went into a stupor and refused to eat. 25 hr later, it ate a little, then died in the night. The author feels that the efficacy of alcohol injections in strychnine poisoning is demonstrated by these experiences, and feels that alcohol may be considered as an antidote to the poison, or as a powerful sedative, the action of which on the CNS is antagonistic to strychnine.

550. Hamilton, R.

INJECTION OF AMMONIA INTO THE VEINS AS A MEANS OF RESUSCITATION IN ALCOHOLIC AND NARCOTIC POISONING.

Lancet (London), 2: 157-158 (1 ref.),

1879.

E – general – case hist. – DC (antidotal) – drug-dep. humans – blood comp., sites, lymph – cardiovasc. – stimulants – *CAAAL-0 A-0769.

The case of a female alcoholic found in a state of comatose stupor is described. Galvanism and the stomach pump having been tried without effect, 10 drops of ammonia iv were given, and, "The effect was striking. In a few moments she moved slightly—an uneasy, restless motion, and soon after, on being shaken and spoken to, partly opened her eyes and turned her head. The most marked change was in the pulse and mouth. The former, which was not to be felt just before the operation, could now be detected, and after a time counted, whilst the mucous membrane of the mouth and tongue became almost immediately of a natural colour." The author hypothesizes that, in alcoholic and

narcotic poisoning, the admixture of the components of the blood is put out of balance, due to a deficiency of certain elements (i.e., nitrogen and hydrogen), and that the application of ammonia can thus alleviate the condition.

551. Hammes, E. M., Jr.
 CARBON TETRACHLORIDE AS AN INDUSTRIAL HAZARD: A REPORT OF TWO CASES.
 Journal of Industrial Hygiene and Toxicology (Cambridge), 23(3): 112-117 (7 ref.), 1941.
 E – SEC – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – blood comp., sites, lymph – cardiovasc. – G.I. tract – liver, kidney – anti-infectants – *CAAAL-0 A-0770.

Of the 2 case histories presented, 1 concerns a foreman in a factory who was constantly exposed to carbon tetrachloride vapours. He admitted drinking a "couple of whiskies" and an occasional beer daily. He became ill, and was admitted to hospital, whereas his fellow workers, who had had the same exposure for several years, developed no symptoms of poisoning. Nevertheless, in comparing this case to the second, that of an abstainer, the degree of individual idiosyncrasy appears to be the most significant factor, for, contrary to what might be expected, the total exposure to carbon tetrachloride necessary to cause illness in the first case was enormously greater than in the second.

552. Händel, K.
 RECHTSFRAGEN ZUM THEMA „ARZNEIMITTEL UND VERKEHR“. [Legal questions on the topic "drugs and traffic"].
 Arzneimittelforschung (Aulendorf), 14(8): 915-922 (38 ref.), 1964.
 G – ES – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev. – analg., antipyret. – anesthetics – tranquilizers – *CAAAL-0 A-0044.

The legal question concerning driving impairment due to drug use, and the position of the physician and drug manufacturer, are discussed. Incidences of combined use of alcohol and drugs are mentioned in connection with the following drugs in various dosages: hostacaine, thomapyrin (salicylic acid + phenacetin + caffeine), bellergal (ergotamine tartrate + combination of belladonna alkaloids + phenobarbital), gelonida (codeine phosphate + phenacetin + salicylic acid), spalt (containing caffeine, phenacetin, and antipyrine), pyramidon, miltown, saridon (isopyrin + phenacetin + caffeine), and delta-butazolidin (phenylbutazone + prednisolone). Some defendants were found guilty on the basis of the principle of "acta libera in causa," and some were found not guilty, because they could not have foreseen the adverse effect of the drugs.

553. Händel, K.
 DIE GEGENWÄRTIGE PRAXIS DES VERKEHRSSTRAFRECHTS. [The present practice of driving laws].
 In: Wagner, K., et al., eds. *Handbuch der Verkehrsmedizin: unter Berücksichtigung aller Verkehrswissenschaften*. [Handbook of traffic medicine: with respect to all traffic sciences]. Berlin: Springer, pp. 72-88 (106 ref.), 1968.
 G – general – review – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – analg., antipyret. – antidepressants – autonomic agents – tranquilizers – *CAAAL-0 B-0320.

In part II ("Zusammenwirken von Alkohol und Medikamenten," [The interaction of alcohol and drugs], pp. 80-83) the author discusses the joint action of alcohol and drugs, and the possible effect on driving ability. Some cases are presented in which drivers caused accidents after alcohol and drug ingestion, but were acquitted on the grounds that they could not be expected to know the action of drugs when ingested before or after alcohol. Interaction is mentioned in connection with the following compounds: bellergal (ergotamine tartrate + combination of belladonna alkaloids + phenobarbital), gelonida (codeine phosphate + phenacetin + salicylic acid), spalt (containing caffeine, phenacetin,

and antipyrine), pyramidon, miltown, saridon (isopyrin + phenacetin + caffeine), delta-butazolidin (phenylbutazone + prednisolone), nicotine, covatix, phenothiazines, thomapyrin (salicylic acid + phenacetin + caffeine), optalidon, tryptizol, and dihydroergotamine.

554. Händel, K.

VERSCHREIBUNG ALKOHOLUNVERTRÄGLICHER MEDIKAMENTE. [Prescription of drugs incompatible with alcohol].

Blutalkohol (Hamburg), 6: 201-210 (26 ref.),

1969.

G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – CNS – tranquilizers – *CAAAL-0 B-0937.

The relative legal and ethical positions of physician and patient, with respect to the prescription and use of drugs which can have a dangerous effect in combination with alcohol, and the legal consequences of such combinations are discussed. Some drivers are convicted for impaired driving on the basis of *actio libera in causa*, while others are acquitted on the grounds that they could not have foreseen the consequences of combined use of alcohol with drugs. The author stresses the necessity for and responsibility of the physician to inform his patient of potential hazards and side effects, and recommends that a note to this effect be made in the patient's file. In civil legal proceedings, the physician is not bound by medical ethics or secrecy in protecting himself by revealing the medical file and the warning note contained therein. 2 cases of impaired driving, 1 of which resulted in conviction, due to interactions of alcohol with amytal in 1 case and librium in the other, are mentioned. It is concluded that, if a proper warning is given regarding the possible incompatibility of a drug with alcohol, only the patient can be held responsible for the consequences if this warning is ignored, and no claims can reasonably be made against the physician.

555. Handwerker, J. V., Jr.

GOLF COURSE DERMATITIS.

J.A.M.A. (Chicago), 189(4): 331 (1 ref.),

1964.

E – general – DC (sensit.) – humans – cardiovasc. – senses – skel., muscle, skin – miscellaneous – *CAAAL-11169-C1 A-1284.

A physician describes a personal experience with "golf-course dermatitis". Shortly after taking up golf, he noticed that, after ingestion of alcoholic beverages, whether beer, wine, bourbon or scotch, a mild erythematous reaction occurred, usually involving some portion of the forehead or face and the hands. 1 evening, after 18 holes of golf, the writer experienced an overwhelming reaction, characterized by flushing, and followed rapidly by marked palpitation, laryngeal edema, swelling of the nasal mucosa, conjunctival injection, and a marked feeling of impending disaster. The reaction lasted for 2 1/2-3 hr, and responded to supportive measures. The doctor considered a disulfiram-like reaction and allergy to aspirin as diagnostic possibilities. He then abstained from drinking alcohol for 10 months, during which time no further reaction was experienced. The etiology was clarified by later investigation, which revealed that, on the day of the severe reaction, the golf-course greens had been sprayed with a thiram fungicide.

556. Hansman, F. S., and Marshall, S. V.

ACUTE ALCOHOLIC INTOXICATION AND THE EFFECT OF THE INHALATION OF 5% "CARBOGEN" THEREON.

Med. J. Aust. (Sydney), 47: 857-858 (0 ref.),

1960.

E – exp. – DC (decrease) – DC (unchanged) – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – CNS – liver, kidney – *CAAAL-9753-N12 A-0771.

A test was conducted on a 168 lb man who had conditioned himself to believe that carbogen is an effective sobering agent. 500 ml of Scotch whiskey (40% v/v alcohol, 30.5° underproof)—equivalent

to 17.8 oz—was ingested in 1 hr and 10 min, and the subject was rapidly and severely intoxicated. When the blood alcohol concentration reached 200 mg/100 ml blood, the subject was given air to breathe which he believed to be carbogen. The psychological effect was extraordinary, the subject claiming that “it was wonderful”, and that he felt better. 5% carbogen was then inhaled to the limit of endurance, but it failed to affect the degree of sobriety, either at the time of inhalation or subsequently.

557. Harger, R. N., and Hulpieu, H. R.

THE EFFECT OF CERTAIN DRUGS ON THE METABOLISM OF ETHYL ALCOHOL.

J. Pharmacol. Exp. Ther. (Baltimore), 54: 145 (0 ref.), 1935.
 E – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev.
 – other drug lev. – metab. proc. – respir. – hormones, hormone antag. – indust. intox. –
 *CAAAL-101-A2 A-0772.

The effects of insulin, thyroxin, dinitrophenol, and dinitrocresol on the normal blood alcohol curve was investigated in dogs. Alcohol was administered by stomach tube in doses of 1.5 or 3.0 g/kg, and determinations of blood alcohol made over a period of 12 hr. Insulin and thyroxin had no effect, whereas dinitrophenol (7.5 or 15 mg/kg) and dinitrocresol (7.5 mg/kg) greatly accelerated the disappearance of alcohol. The high doses of the latter two, which were necessary to cause significant acceleration, are probably too toxic to be of practical use in the treatment of acute intoxication.

558. Harris, F. H.

ACUTE CARBON TETRACHLORIDE POISONING.

United States Armed Forces Medical Journal (Washington), 3(7): 1023-1028 (1 ref.), 1952.
 E – general – DC (add., infra-add., unspec. incr.) – humans – other drug lev. – blood comp., sites,
 lymph – G.I. tract – liver, kidney – anti-infectants – *CAAAL-0 A-0773.

78 men were directly or indirectly exposed to carbon tetrachloride fumes for 1-16 hr, 49 of them subsequently developing symptoms of some kind. 15 men were treated for poisoning. A great many of the group had consumed alcohol at a Christmas celebration the night before, and many were suffering from hangovers. Of those with symptoms, 63% had ingested alcohol the night before or more recently. Of those who drank alcohol, 74% had symptoms, whereas, of those who had drunk no alcohol, 50% had symptoms. Of the 15 cases of poisoning, all except 1 had drunk alcoholic beverages, and this 1 was the least affected. The 6 most severe cases of poisoning all involved “steady drinkers” who had consumed large quantities of alcohol the night before.

559. Hartmann, A.

UNTERSUCHUNGEN ÜBER DIE ART DER ERNÜCHTERUNG NACH CARDIAZOL- UND CORAMINGABEN AM ALKOHOLBERAUSCHTEN TIER. [Investigations of the kind of sobering effects after administration of cardiazol and coramine in the intoxicated animal].

Dissertation, Medical Faculty of the University of Würzburg, West Germany, 37 pp. (27 ref.), 1953.

G – exp. cont. – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – other drug lev. – CNS – liver, kidney – respir. – stimulants – *CAAAL-0 A-0774.

A series of controlled experiments were carried out (4 rats/test). Alcohol (25%) was administered po in a dose of 0.02 cc/kg, followed 30 min later by 0.025 mg/kg cardiazole sc, 0.15 mg/kg coramine, or cardiazole plus coramine. The animals were killed after 1 hr, dissected, and the alcohol level determined in the individual organs of the body. The obtained values were plotted graphically and tabulated. It was found that the analeptics shifted the alcohol level in the individual organs. The concentration became lower in the liver and lungs. In the kidneys the reverse was the case, conceivably because of the better blood flow caused by the drugs. The alcohol concentration of the brain was not

affected. In view of the complexities of the reaction mechanisms involved, the author suggests that the clinically observed rapid sobering effect should not be viewed as genuine but apparent.

560. Haskins, H. D.

STUDIES ON THE ANTAGONISTIC ACTION OF DRUGS.

Amer. J. Med. Sci. (Philadelphia), 126: 1036-1047 (5 ref.), 1903.

E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – in vitro – CNS – nerv. syst. – respir. – stimulants – *CAAAL-0 A-0775.

The effect of strychnine in alcohol poisoning was investigated in guinea pigs and cats (1 pair/test). 40% alcohol (total of 40 cc/kg in guinea pigs, 20 cc/kg in cats) was given in several doses. 1 of each pair received strychnine sulphate (total of 0.15 mg/kg for guinea pigs, 0.1-0.15 mg/kg for cats) in a 1:20,000 sol sc in several doses. The results showed that strychnine does not seem to affect the pulse when given simultaneously with alcohol. In some cases, strychnine seemed to counteract in part the depressing effect of acute alcoholic poisoning on the respiration, and thus prolong life. It does not interfere materially with the narcosis from acute poisoning with alcohol, but, apparently, does delay the onset of paralytic symptoms. Although in no case was strychnine able to completely remove the effects of the large alcohol doses, it is considered probable that fairly strong therapeutic doses of strychnine would be effective in treating poisoning, from even lethal alcohol doses.

561. Hatfield, G. K.

MODIFICATION OF DRUG ACTION BY REPEATED ETHANOL TREATMENT IN THE RAT.

Ph.D. Thesis, School of Graduate Studies of Purdue University, Lafayette, Indiana, U.S.A., 61 pp. (69 ref.), 1966.

E – exp. cont. – exp. comp. – cross-tol. – DC (decrease) – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – blood lev. – mot. perform. – species or sex diff. – CNS – liver, kidney – metab. proc. – anti-infectants – barbiturates – musculoskel. agents – tranquilizers – *CAAAL-0 B-0321.

The interaction of ethanol and various CNS depressants was investigated in male and female rats. The animals received 2.5 g/kg ethanol/day for 4, 10, or 15 days ip. 24 hr after the last ethanol dose, 200 mg/kg barbital, 4.0 g/kg ethanol, 80 mg/kg hexobarbital, 250 mg/kg meprobamate in 2% acacia, 30 mg/kg pentobarbital, 125 mg/kg phenobarbital, or 80 mg/kg zoxazolamine in 2% acacia, were given ip. In male rats, no cross-tolerance between ethanol and the other CNS depressants was produced (as shown by loss of righting reflex or alteration in plasma drug levels); cross tolerance was observed in female rats between ethanol and hexobarbital, pentobarbital, zoxazolamine, and meprobamate, but not between ethanol and barbital or phenobarbital. The tolerance in the female was shown not to be due to an increased rate of drug metabolism, and is probably due to a CNS cellular tolerance to the drugs.

562. Hauschild, F.

ZUR PHARMAKOLOGIE DES 1-PHENYL-2-METHYLAMINOPROPANS (PERVITIN).

[The pharmacology of 1-phenyl-2-methylaminopropane (pervitin)].

Naunyn Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 191: 465-481 (29 ref.), 1939.

G – SEC – exp. – DC (decrease) – mammals – acute admin. – in vivo – CNS – amphetamines – *CAAAL-0 A-0776.

Pervitin was tested on rats, mice, cats, and rabbits, and was found to have a stimulating and excitatory effect and to increase blood pressure. It showed antagonistic effects with sedatives and hypnotics, and shortened sleeping time and narcosis. The interaction of pervitin and alcohol was investigated in

rabbits. The time interval between the injection of alcohol and the return of the animal to normal running behaviour was used as criterion. 30% alcohol was given ip, 0.4 g/100 g, and pervitin administered immediately afterwards, 0.3 mg/100 g ip. The median time for return to normal behaviour was 258 min after alcohol alone, and 157 min after alcohol plus pervitin.

563. Hauschild, F.
TÖDLICHE KALKSTICKSTOFFVERGIFTUNG UND DIE FRAGE DES GESTÖRTEN
ALKOHOLABBAUES. [Lethal calcium nitrate poisoning and the question of disturbed alcohol
metabolism].
Sammlung von Vergiftungsfällen (Berlin), 14: 311-320 (28 ref.), 1952-54.
G – general – DC (sensit.) – med.-leg. – post-mort. – humans – cardiovasc. – miscellaneous –
*CAAAL-6647-E3 A-1399.

A case is reported concerning an agricultural labourer who was exposed to calcium nitrate fertilizer, and later complained of constant thirst, a burning sensation in the throat and larynx, an urgent desire to urinate, and strong fatigue. 18 days later, a physician examined the man, diagnosed calcium nitrate poisoning, and ordered rest. During the following 17 days until hospitalization, the man's condition deteriorated, and a stiffening developed in the left leg. A few days after admission to hospital he died. An autopsy revealed an embolism in the left leg, and thrombosis of the left iliac artery. It was concluded that death resulted from complications of diabetes. Due to the denial of insurance compensation to the man's wife for this non-occupational cause of death, the decision was appealed. During the appeal hearing, expert testimony submitted that the deceased had consumed alcohol on the day of exposure, and that this factor contributed to the fatal outcome of the poisoning. 10 similar cases of lethal calcium nitrate poisoning were presented, following which the appeal was granted, and the combined toxic effect accepted as the cause of death. The chemistry and effects of calcium nitrate are described, and a comparison with the effects of disulfiram is made.

564. Hazleton, L. W., and Hellerman, R. C.
THE INFLUENCE OF VEHICLES ON THE ACTION OF DRUGS.
J. Amer. Pharm. Ass. (Washington), 35(6): 161-168 (11 ref.), 1946.
E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute
admin. – in vivo – dose resp. – blood lev. – absorp., distrib., stor. – G.I. tract – barbiturates –
stimulants – *CAAAL-4731-D3 A-0777.

The influence of such vehicle ingredients as sucrose, dextrose, glycerin, propylene glycol, and alcohol on the gastrointestinal absorption of sodium barbital and metrazol was studied in mice. The median effective dose (ED₅₀) for alcohol was established and administered ip or po in 10, 25, or 50% sol, in combination with 30 or 50 mg/kg sodium pentobarbital or 20, 60, 100, or 150 mg/kg metrazol. In a second series of tests, the compounds were administered separately, one iv and the other po, and vice versa. The ED₅₀ of alcohol in 10% sol was administered with 30 or 50 mg/kg pentobarbital, and with 20 or 150 mg/kg metrazol. The results showed that alcohol does not interfere with the gastrointestinal absorption of pentobarbital or metrazol. Po administration of alcohol in concentrations of 10% or more increases the action of pentobarbital, and decreases that of metrazol; it is a systemic action independent of any local effect. Iv alcohol administration exerts a systemic action which interferes with the absorption of these 2 drugs; this does not occur after po administration. Results concerning other vehicles are also discussed.

565. Heck, K., Mallach, H. J., Mayer, B., and Mayer, K.
ÜBER DIE GEMEINSAME WIRKUNG VON ALKOHOL UND DMSO BEIM
MENSCHEN. [On the combined action of alcohol and DMSO in man].
Med. Welt (Stuttgart), 17(37): 1963-1975 (25 ref.), 1966.
G – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – absorp.,
distrib., stor. – senses – *CAAAL-0 B-0322.

The interaction of alcohol (0.75 g/kg po) and dimethylsulfoxide (DMSO—50 mg/kg sc) was investigated in healthy human subjects. 15 subjects received DMSO, followed 60 min later by alcohol. 15 received both substances simultaneously, and 16 received alcohol only. The results showed that DMSO does not cause an increase of alcohol absorption or an elevated blood alcohol level. Testing of concentration ability and reaction time, using acoustic and visual stimuli, showed that impairment was greater after DMSO-alcohol, than after alcohol alone; impairment was slightly greater in the group receiving alcohol 60 min after DMSO, than in the group given the 2 substances simultaneously.

566. Heidenreich, O.

DIE DIURETISCHE WIRKUNG VON COFFEIN UND ALKOHOL BEI NORMALEN UND HYPOPHYSEKТОMIERTEN HUNDEN. [The diuretic action of caffeine and alcohol in normal and hypophysectomized dogs].

Arzneimittelforschung (Aulendorf), 7(7): 439-442 (24 ref.), 1957.

G – ES – exp. cont. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – other drug lev. – acid-base, blood pH, elect. – glands – liver, kidney – stimulants – *CAAAL-8246-B2 A-0778.

The diuretic effect of caffeine and alcohol was investigated after separate and simultaneous oral application in normal and hypophysectomized female dogs. 5 dogs were episiotomized and catheterized hourly. Urine vol, and chloride, potassium, and sodium elimination were measured. After 1 hr, the dogs received by stomach tube: (1) caffeine sodium salicylate, 40 mg/kg in 20 ml of water; (2) absolute alcohol, 1.5 ml/kg in 33% sol; and (3), (1) and (2) in combination. It was found that simultaneous application of caffeine and alcohol induced a marked increase of diuresis. The amount of water excreted was twice as high as might be expected if the effect of both drugs were additive. The concentration of electrolytes was 70% lower than previously. When caffeine and alcohol were applied simultaneously to hypophysectomized dogs, the urinary excretion was in the same range of magnitude as after separate application of alcohol. It is concluded that caffeine induces the release of an antidiuretic pituitary hormone in dogs, thus reducing the diuretic effect of caffeine. If the release of the hormone is prevented by alcohol, the diuretic effect of caffeine will occur.

567. Heilner, E.

VERSUCH EINES INDIREKTEN FERMENTNACHWEISES (DURCH ALKOHOLZUFUHR); ZUGLEICH EIN BEITRAG ZUR FRAGE DER UEBEREMPFINDLICHKEIT. [Test of an indirect fermentation (through alcohol induction); also a contribution to the question of hypersensitivity].

Munchen. Med. Wschr. (Munich), 55(49): 2521-2524 (56 ref.), 1908.

G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – anti-infectants – *CAAAL-0 A-0779.

Rabbits were checked for 3 days for urinary excretion, temperature, and weight. On the 4th day, they received 275-294 cc horse serum sc, with 24.45-25.31 g albumin. 3 groups received, immediately after the serum, 10 cc 96% alcohol in 35 cc water by stomach tube. 1 of these groups received 0.1 cc quinine in 10 cc water sc on the 6th day. In 1 group, the above amount of alcohol was again administered on the 5th and 6th days. The experiments were terminated after 8 days. The results showed that alcohol significantly accelerated the decomposition of the foreign albumin circulating in the blood, either by promoting the formation of ferment, or by increasing the effects of the ferment.

568. Hein, J., and Stecher, W.

ZUR INH-THERAPIE. [INH therapy].

Z. Tuberk. (Leipzig), 101(1-2): 83-98 (84 ref.), 1952.

G – SEC – exp. – general – DC (sensit.) – humans – chronic admin. – in vivo – cardiovasc. – anti-infectants – *CAAAL-0 A-1451.

The effects of isonicotinic acid hydrazide (INH) as a tuberculostatic agent are described, and the drug is compared with other antituberculous agents currently in use. The observations of a 6-month clinical study conducted by the author, employing a daily INH dosage of 4-6 mg/kg, are reported. It was found that INH is effective during the first 2 months of treatment, after which the number of recidivists increases. Extended use sometimes results in a worsening of the condition. Although INH is well-tolerated, certain side-effects, such as dizziness, a fall in body temperature, and intolerance to alcohol, are sometimes seen. 1 patient receiving INH treatment fell into a coma after drinking a moderate amount of alcohol, and developed a slowly-decreasing hypertonia. Other patients also complained of a revulsion towards beer. It is concluded that INH is the most tolerable tuberculostatic agent thus far developed, but its therapeutic value should not be overestimated.

569. Heinrichs, K. M.

UNTERSUCHUNGEN ÜBER DIE EINWIRKUNG DER TRANQUILLANTIEN „DOMINAL-FORTE“, „MILTAUN“, „SOPRINTIN“, „VEROPHEN“, UND DES STIMULANS „TRADON“ BEI GLEICHZEITIGEM ALKOHOLGENUSS. [Investigations of the effect of the tranquilizers “dominal-forte”, “miltown”, “soprintin”, “verophene” and of the stimulant “tradon” taken simultaneously with alcohol].

Dissertation, Medical Faculty of the University of Heidelberg, West Germany, 60 pp. (6 ref.),

1961.

G – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – CNS – metab. proc. – sed., hypnot. – stimulants – tranquilizers – *CAAAL-0 A-0780.

A number of tests were carried out in healthy subjects with dominal-forte, a thiophenylpyridylamine derivative (2 x 40 mg), miltown (400 mg), soprintin, a phenothiazine derivative (3 tablets), verophene, a propylamine phenothiazine derivative (3 x 0.025 g), and tradon, 5-phenyl-2-imino-4-oxo-oxazolidin (3 tablets), in combination with alcohol (70-120 g) under controlled conditions. The results were as follows: dominal-forte showed varying reactions—one subject had slight euphoria and no fatigue, the other manifested appreciable symptoms of fatigue and intoxication. Miltown did not potentiate the effect of alcohol; rather, improved performance was shown in some tests, as compared with controls. No marked sedation was noted. Soprintin potentiated the alcohol effect. Sedation was manifested in the subjects towards the end of the test. Tradon had an appreciable sedating action. None of the substances had any effect on alcohol metabolism.

570. Henley, K. S., and Scholz, R.

ETHANOL: DRUG, “HEPATOTOXIN,” AND MODIFIER OF INTERMEDIARY METABOLISM.

Med. Clin. N. Amer. (Philadelphia), 53(6): 1413-1423 (41 ref.),

1969.

E – SEC – general – review – cross-tol. – DC (add., infra-add., unspec. incr.) – humans – mammals – blood lev. – liver, kidney – metab. proc. – barbiturates – *CAAAL-0 B-0523.

The modification by alcohol of its own metabolism and that of other compounds is discussed. Ethanol is known to be an inducer of microsomal drug-metabolizing systems, associated with a proliferation of the endoplasmic reticulum in hepatic parenchymal cells. It is known clinically that many alcoholics, when sober, are more resistant to drugs, and require higher doses of barbiturates for a given effect; when drunk, alcoholics are more susceptible to barbiturates, with possible lethal consequences. In vitro experiments have shown that ethanol inhibits the activities of microsomal enzymes capable of metabolizing phenobarbital. If phenobarbital is used to induce the microsomal drug-metabolizing system, lipid accumulation in the liver from ethanol ingestion is prevented, not by lowering the blood level of ethanol, but by lowering the lactate levels. This suggests that more ethanol is being oxidized by a microsomal pathway, and that less of the NADH generated is removed by the lactic dehydrogenase reaction.

571. Hermanns, A.

ÜBER ATYPISCHEN VERLAUF VON SCHLAFMITTELVERGIFTUNGEN BEI GLEICHZEITIGEM ALKOHOLGENUSS. [On the atypical course of sedative poisoning when combined with alcohol intake].

Dissertation, Medical Faculty of the University of Bonn, West Germany, 19 pp. (24 ref.), 1954.
G – stat. surv. – DC (supra-add. incr.) – post-mort. – humans – blood lev. – barbiturates – *CAAAL-0
A-0781.

7 detailed case histories are presented in which death occurred after sublethal doses of barbiturates, concomitant with a moderate to high blood alcohol level (BAL). Case 1: 2.8 g phanodorm po; BAL 2.2°/oo; death after 3 hr. Case 2: undisclosed but small amount of barbiturates; BAL 0.15°/oo; dead on arrival. Case 3: considerable dose of phanodorm; BAL 2.2°/oo; death after 4 hr. Case 4: found dead with phanodorm present; BAL 0.2°/oo. Case 5: found dead; small amount of barbiturates; BAL 2.6°/oo. Case 6: found dead; considerable amount of barbiturates; BAL 0.9°/oo. Case 7: found dead; phanodorm and evipan present; BAL 2.0°/oo. In all cases, the post-mortem revealed no other cause of death besides the drug-alcohol interaction. The amounts of barbiturates determined were small except in cases 3 and 6, and, according to known lethal doses, were not sufficiently large to cause death. The interaction was considered to be potentiative, rather than synergistic.

572. Hernández-Peón, R., Goldberg, L., and Rojas-Ramirez, J. A.

PHYSIOLOGY AND PSYCHOSOMATIC MEDICINE: NEUROPHYSIOLOGICAL MODELS OF EMOTIONAL BEHAVIOR AND OF ACTION OF PSYCHOTROPIC DRUGS.

In: Pletscher, Alfred and Marino, A., eds. *Psychotropic Drugs in Internal Medicine*. Proceedings of an international symposium, Baia Domisia, Italy, May 3-4, 1968, sponsored by the Instituto di farmacologia dell'Universita di Napoli. Amsterdam: Excerpta Medica Foundation, pp. 16-46 (16 ref.) @ 1969.

E – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – mot. perform. – psychol. perform. – CNS – tranquilizers – *CAAAL-0 B-0938.

Various aspects of neurophysiological mechanisms in psychosomatic disorders and psychotropic drug action are discussed. The separate and combined effects of ethanol (0.3 g/kg ip, or 4 µg in 0.02% (v/v) intracerebrally), chlordiazepoxide (0.3 mg/kg ip), and diazepam (0.15 mg/kg ip) were studied in cats by recording background activity and responses to local and mediated evoked potentials by means of chronically implanted electrodes and micro-cannulae for intracerebral drug administration. Changes were recorded in the wakefulness-sleep spectrum, in reticular formation, in hypothalamic structures, on arousal, on sensory transmission and filtering, on mnesic function in the entorhinal cortex, on the limbic emotional system, and on cortical and subcortical reactivity in general. Chlordinazepoxide counteracted all alcohol effects on the structures studied. The alcohol-induced shift to prolonged sleep periods was counteracted, and wakefulness-sleep patterns were normalized. Alcohol-impaired behaviour was antagonized, the animals staying alert and relaxed, and some of the prolonged after-effects of alcohol, which in the hypothalamus and cerebral cortex lasted up to 24 hr, were prevented. Most of the alcohol effects studied, from behaviour to effects on the reticular formation and the entorhinal cortex, were enhanced by diazepam; counteracted, however, were the effects of alcohol on the amygdaloid limbic complex and the wakefulness-sleep spectrum.

573. Herr, F., Stewart, J., and Charest, M. -P.

TRANQUILIZERS AND ANTIDEPRESSANTS: A PHARMACOLOGICAL COMPARISON. Arch. Int. Pharmacodyn. (Gand), 134(3-4): 328-342 (13 ref.), 1961.

E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – tranquilizers – *CAAAL-0 A-1400.

In an attempt to discover reliable procedures in animals for identifying compounds of the imipramine type, the pharmacological actions of a new compound, amitriptyline, were investigated and compared to those of imipramine, chlorpromazine, and chlorprothixene. Acute toxicities in mice, potentiation of subnarcotic doses of alcohol in mice, potentiation of hexobarbital narcosis in mice, effects on body temperature of mice, spontaneous motility in rats, ataxia in rats, the runway test in rats, avoidance performance in rats, and learning of avoidance in rats, were investigated. Different doses of the 4 compounds were administered ip to mice (10 mice/drug dose) 30 min prior to 4 g/kg ethanol ip. This alcohol dose caused a loss of righting reflex (LRR) in a max of 5% of non-pretreated animals. Strength of potentiation was determined by the incidence of LRR, and results were calculated in "all or none" terms. All compounds enhanced the effects of alcohol; however, the effective dose range for imipramine and amitriptyline was much higher than that for chlorpromazine and chlorprothixene. Although amitriptyline was more powerful than imipramine, it could not be grouped with chlorpromazine and chlorprothixene by its effectiveness on the test.

574. Hess, J.
 ÜBER DIE WECKWIRKUNG VERSCHIEDENER ANALEPTICA BEI DER ALKOHOL-
 UND CHLORALLOSENARKOSE. [The stimulative effect of different analeptics in alcohol and
 chloral narcosis].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 197:
 204-209 (7 ref.), 1941.
 G – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals
 – acute admin. – in vivo – dose resp. – CNS – amphetamines – analeptics – stimulants – *CAAAL-0
 A-0782.

The antagonistic action of cardiazole, coramine, pervitin, and cycliton on alcohol narcosis was studied in rats. The optimal doses with the best arousal effect were 2.5 mg/100 g for cardiazole, and 15 mg/100 g for coramine. 30 mg/100 g of coramine had a synergistic effect on the alcohol, and prolonged the narcosis. Pervitin (25, 50, 75, or 100 µg/100 g body wt) shortened the narcosis only slightly; cycliton (3, 6, 12, or 24 mg/100 g body wt) had no arousal effect. None of the above-mentioned drugs had an antagonistic effect on the chloral narcosis.

575. Hesse, E.
 ÜBER DIE CYANAMIDWIRKUNG. I. MITTEILUNG. [The action of cyanamide. I].
 Z. Ges. Exp. Med. (Berlin), 25: 321-344 (24 ref.), 1921.
 G – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (sensit.) – mammals – other org. – acute
 admin. – in vivo – cardiovasc. – G.I. tract – liver, kidney – respir. – *CAAAL-0 A-1401.

Experiments were performed on dogs, cats, rabbits, and frogs, to study the toxic effects of cyanamide, alone and in combination with alcohol and other drugs, on the heart, circulation, respiration, body temperature, kidneys, and intestines. Combined effects of alcohol and cyanamide were investigated in rabbits and frogs. 1 rabbit was given 5 g 96% alcohol (sol in 50 cc water), 6 hr prior to successive iv doses of 0.1 g cyanamide, up to a total of 0.4 g. Another rabbit received 0.3 g cyanamide iv, followed by the above alcohol dose. In other tests on rabbits, 0.2 and 0.1 g cyanamide were combined with various doses of ethanol, other alcohols, and sedatives and hypnotics. It was found that toxic heart damage induced by alcohol was unaffected by cyanamide. Blood pressure, however, was more strongly lowered under the combined effect. Body temperature was significantly decreased, while no increased effect was noted in respiration and pulse. Tests on frogs indicated no enhancement of effects, whether alcohol was given before or after cyanamide. It is concluded that cyanamide promotes the action of alcohol and a large range of other substances; conversely, however, alcohol does not potentiate the action of cyanamide. A case of intolerance to alcohol is reported in a man who took 150 mg cyanamide po early in the morning, and, later in the evening of the same day, drank a glass of beer. A headache, nausea, and rash on the face and neck persisted for about 6 hr.

576. Hesse, E.

ÜBER DIE CYANAMIDWIRKUNG. II. MITTEILUNG. [The action of cyanamide. II].

Z. Ges. Exp. Med. (Berlin), 26: 337-351 (17 ref.),

1922.

G – SEC – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – in vitro – other drug lev. – absorp., distrib., stor. – CNS – liver, kidney – *CAAAL-0

A-1402.

The combined effects of cyanamide and its derivatives, dimethyl-, diethyl-, diamyl-, and methyl-phenylcyanamide, with a wide range of substances, were investigated in dogs, rabbits, and frogs. In 1 experiment, rabbits were pretreated with 0.2 g/kg cyanamide, followed 1 hr later with either 2 g 96% ethanol in 50 cc water/kg body wt or methanol. When body temperature changes were detected, the animals were sacrificed, and alcohol concentrations in the brain and spine were determined. The amount of alcohol present in the CNS of pretreated animals was greater than in controls. No significant difference was observed between test and control groups in vitro. The cyanamide derivatives showed no potentiation of the alcohol effect. It is concluded that cyanamide increases the concentration of certain substances in specific organs, such as the brain or kidneys, probably due to the solubility of cyanamide in ether or lipids. An increase of alcohol in the CNS can be the cause of previously-observed temperature loss, following combined cyanamide-alcohol administration.

577. Hessling, B.

UEBER EINIGE ANTIDOTE DES STRYCHNIN. [Some antidotes to strychnine].

Dissertation, Medical Faculty of the University of Göttingen, Germany, 40 pp. (7 ref.), 1877.

G – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – CNS – skel., muscle, skin – stimulants – *CAAAL-0

A-0783.

A series of tests were carried out with potassium bromide-chloral hydrate (in combination), alcohol, and physostigmine, to determine their antidotal properties to strychnine. Absolute alcohol (4 g) in a 50% sol in water injected into a rabbit (1200 g wt), followed by 0.7 mg strychnine and 1 g alcohol 20 min later, evoked spasms (tetanus) of 30 sec duration. The animal made a moderate recovery 3 1/2 hr later. 5.5 g absolute alcohol given to another rabbit of the same wt, followed by 0.7 mg strychnine and 1 g alcohol 45 min later, gave somewhat better results. Best results were obtained with 5 g alcohol (absolute) administered to a 1000 g rabbit, followed by 0.8 mg strychnine with 1 g alcohol 30 min later. The rabbit was fully recovered after 75 min sleep. The author concludes that alcohol may be regarded as an effective antidote to smaller quantities of strychnine, but it is most uncertain for lethal doses.

578. Heubner, W., and Hallermann, W.

ZUR TOXICITÄT DES PYRAMIDONS. [The toxicity of pyramidon].

Arch. Toxik. (Berlin), 15: 157-158 (0 ref.),

1954.

G – general – exp. – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – mammals – acute admin. – in vivo – dose resp. – analg., antipyret. – *CAAAL-0

A-0784.

Case material is presented of a girl poisoned by the combination of pyramidon (aminophenazone) and alcohol given to her in a bar by her escort. Shortly thereafter, the girl's body was found lying on the curb of the street. An examination of the case disclosed that death occurred 15-20 min after ingestion of the pyramidon-containing liquor. The young man was found guilty of murder. Laboratory tests revealed that it is easy to mix a tolerably-tasting drink containing, in addition to 50% alcohol, so much pyramidon that, with 2 or 3 sips, 15 g of the drug can be ingested. Comparable oral doses were lethal in rats. The experiments with rats demonstrated also that dosages which showed no observable symptoms and lethal dosages were very close. It was concluded that simultaneous intake of alcohol increases the pyramidon toxicity considerably.

579. Higgins, J. A., and McGuigan, H. A.
 THE INFLUENCE OF CAFFEINE ON THE EFFECTS OF ACETANILID.
 J. Pharmacol. Exp. Ther. (Baltimore), 49: 466-478 (11 ref.), 1933.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – cardiovasc. – respir. – analg., antipyret. – *CAAAL-0 A-0785.

The action of acetanilide on the heart was investigated in dogs, rabbits, and mice. To test the toxicity of acetanilide in an alcohol sol, rabbits were given 92-133% of the fatal combination of fractions of the lethal doses of alcohol (lethal dose—8 cc 95% alcohol/kg) and acetanilide (lethal dose—1.5 g/kg), 102% of the combined toxic dose thus containing 1.35 g/kg acetanilide plus 1.0 cc/kg ethanol. The results showed that the combined effect of alcohol and acetanilide is additive; rabbits which got more than 100% of the combined dose died, whereas those who received less survived.

580. Hillbom, M., and Pikkarainen, P.
 ETHANOL INHIBITION OF SORBITOL OXIDATION IN LIVER SLICES OF PROPYL
 THIOURACIL-TREATED AND CONTROL RATS.
 Life Sci. (Oxford), 7(13): 713-720 (16 ref.), 1968.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. – in vitro – glands – liver, kidney – metab. proc. – hormones, hormone antag. – *CAAAL-0 B-0323.

Male rats given a choice between tap water and 10% alcohol, were divided into 2 groups of 7 rats each. The first group was intubated daily with 5 mg of a 0.5% propylthiouracil (PTU) sol, and the second group with 1 ml tap water/100 g. After 3 months, the rats were killed and slices from each liver incubated for 3 hr with 1.5 mM lactate, 11.0 mM glucose, and 6.9 mM sorbitol. One flask of each set also contained 54.0 mM ethanol. The oxidation rate of sorbitol in liver slices from PTU-treated rats was not significantly different from the untreated. The presence of ethanol significantly inhibited sorbitol oxidation in both groups.

581. Himwich, H. E., Sykowski, P., and Fazekas, J. F.
 THE EFFECTS OF ALCOHOL AND PENTOBARBITAL ON METABOLISM OF
 EXCISED CEREBRAL TISSUES OF ADULT AND INFANT RATS.
 Amer. J. Physiol. (Bethesda), 129: 382-383 (0 ref.), 1940.
 E – abst. – exp. cont. – mammals – in vitro – CNS – metab. proc. – barbiturates – *CAAAL-2505-B2 A-0786.

The effects of alcohol and pentobarbital on the oxygen consumption of the adult and infant rat brain were examined. The relative rate of metabolism of the various parts of the adult brain was found to be in the following descending order: cortex, brain stem, cerebellum, medulla. In the presence of 6% alcohol, absolute depression occurred in the same order, and the percentage depression was approximately the same (38% for cortex, cerebellum and medulla, but less, 19%, for the brain stem). The infant brain seemed to be more resistant to metabolic depression.

582. Hine, C. H., and Turkel, H. W.
 RESEARCH OF THE SCIENTIFIC LITERATURE AND REPORTS ON THE EFFECTS ON
 MAN OF ALCOHOL ALONE AND IN COMBINATION WITH OTHER DRUGS.
 U.S.A.F. Arctic Aeromedical Laboratory, Ft. Wainwright, Alaska, Report AAL-TR-63-22, 157 pp. (244 ref.) July, 1966.
 E – exp. – review – congen. stud. – DC (antidotal) – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – DC (sensit.) – humans – drug-dep. humans – in vivo – in vitro – dose resp. – blood lev. – other drug lev. – mot. perform. – psychol. perform. – acid-base, blood pH, elect. – blood comp., sites, lymph – cardiovasc. – CNS – liver, kidney – metab. proc. – respir. – senses – elect., water-bal. agents – stimulants – tranquilizers – *CAAAL-0 B-0324.

Reviewed is the medical and scientific literature regarding the effects of alcohol alone and in combination with other drugs on the behaviour of animals and man in low environmental temperatures. Special attention is given to pharmacology, physiology, effects on the nervous system, and behaviour following acute or repeated intake of alcohol.

583. Hoenig, V., Brodanová, M., and Kordač, V.

EFFECT OF ETHANOL ON IRON TOLERANCE AND ENDOGENOUS SERUM IRON IN LIVER CIRRHOSIS.

Scand. J. Gastroent. (Oslo), 3: 334-338 (39 ref.),

1968.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – liver, kidney – nutritive agents – *CAAAL-13743 B-0524.

To determine whether increased iron deposits found in livers of chronic alcoholics were due to increased gastro-intestinal iron absorption because of chronic ethanol intake, 8 normal and 16 cirrhotic patients were studied for: the effect of 60 g ethanol in 40% sol po on oral and iv iron tolerance curves (produced by 1200 mg iron gluconate), and on the endogenous serum iron level. Results of iron level measurements indicate that, in normal subjects, ethanol had no significant effect on iron tolerance curves, but, in cirrhotic patients, the curve was significantly elevated. Ethanol did not affect the endogenous serum iron level, nor did it affect the serum clearance of exogenous iron. There was, however, an increase in the cirrhotic oral iron tolerance curve after ethanol, indicating that the ethanol potentiated the already increased iron absorption found in liver cirrhosis. The elevated curve could be normalized by 1500 mg pancreatin po, and lowered by 90 units of pancreozymin iv.

584. Hofacker, U.

DER EINFLUSS VON ANALGETISCH WIRKSAMEN MISCHPRÄPARATEN AUF DEN ALKOHOLSTOFFWECHSEL (-ABBAU). [The influence of mixed preparations of analgetics upon the metabolism of alcohol].

Dissertation, Medical Faculty of the University of Mainz, West Germany, 30 pp. (49 ref.),

1962.

G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – analg., antipyret. – nutritive agents – *CAAAL-0 A-0737.

The acetaldehyde and pyruvic acid values in the blood serum of 165 albino rats were determined by the enzymatic method; the rats received no treatment, alcohol, or alcohol plus a drug, during experiments lasting 3 days. The drugs used were: thornapyrin (28.60-150 mg/kg/day), eumed (28.-60-150 mg/kg/day, by stomach tube), saridon (24.60-36.84 mg/kg/day, by stomach tube), and cibalgil (15.20-22.80 mg/kg/day im). 1.5 g/kg alcohol was given as 30% sol by stomach tube. The drugs increased the acetaldehyde level, but not significantly. Thomapyrin caused the highest levels, both alone and in combination with alcohol. It is concluded that simultaneous ingestion of the drugs and alcohol would impair driving ability.

585. Hoffer, A.

LACK OF POTENTIATION OF ALCOHOLIC EXCITEMENT BY METHYPRYLON (NOLUDAR).

Canad. Med. Ass. J. (Toronto), 79(3): 191 and 217 (0 ref.),

1958.

E – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – CNS – sed., hypnot. – *CAAAL-8775-J1 A-0788.

The purported ability of methyprylon to potentiate the effects of alcohol was investigated in 4 experiments, each involving 6 human subjects. In each test, 3 subjects received placebo, and 3 received methyprylon po in the following dosages: 100 mg at 2:30 pm, 50 mg at 5:00 pm, and 50 mg at 8:00 pm. The design was randomized, double blind. At 9:00 pm all subjects received 6 1/2 oz Canadian

standard rye whiskey in water. The results showed that methyprylon does not potentiate the excitement and social disinhibition produced by alcohol.

586. Hoffer, A., and Osmond, H.

CONCERNING AN ETIOLOGICAL FACTOR IN ALCOHOLISM: THE POSSIBLE ROLE OF ADRENOCROME METABOLISM.

Quart. J. Stud. Alcohol (New Haven), 20: 750-756 (10 ref.), 1959.

E – SEC – general – cross-tol. – drug-dep. humans – blood lev. – psychol. perform. – glands – hallucinogens – *CAAAL-8718-L3 A-1403.

The authors propose that an oxidized derivative of epinephrine, such as adrenalin or something like it, neutralizes tension due to increased epinephrine, and that tension is thus proportional to the ratio of epinephrine to adrenochrome (adrenochrome metabolites or analogues)—a high ratio is associated with tension, and a low ratio with relaxation. Because of inadequate synthesis or too rapid metabolism of adrenochrome, the chronic excessive tension observed in alcoholics, as well as their inadequate response to LSD, may be due to an imbalance between the amounts of epinephrine and adrenochrome present, an imbalance which can be temporarily corrected by injected adrenochrome. Hence, alcohol is a substitute for the missing adrenochrome. In therapeutic trials with LSD, it was found that alcoholics needed larger quantities of LSD than normal subjects, to obtain an equivalent experience. In 4 normal subjects, 100 μ g LSD increased plasma adrenochrome from a mean of 63 to 197 μ g/l, whereas, in 5 alcoholics given 200 μ g LSD, the increase in adrenochrome was from a mean of 59 to 125 μ g/l. If adrenochrome or adrenolutin was given iv to the alcoholics, tension disappeared rapidly, and a normal or even prolonged LSD experience with smaller LSD doses soon followed.

587. Hoffer, A.

LACK OF POTENTIATION BY CHLORDIAZEPOXIDE (LIBRIUM) OF DEPRESSION OR EXCITATION DUE TO ALCOHOL.

Canad. Med. Ass. J. (Toronto), 87: 920-921 (3 ref.), 1962.

E – exp. cont. – DC (add., infra-add, unspec. incr.) – DC (unchanged) – humans – acute admin. – in vivo – CNS – tranquilizers – *CAAAL-10179-D1 A-0789.

To investigate the interaction of librium and alcohol, 4 double-blind, randomized-design experiments were conducted on human subjects (6/test; 4 subjects in the last test). In each test, 3 subjects received placebo and 3 received 10 mg librium po at 8:30 am, 1:30 pm, and 6:30 pm. At 8:00, 9:00, and 10:00 pm, all subjects were given 2 oz of Canadian rye whiskey. It was concluded that, in this recommended daily dose, librium did not potentiate what was considered a social quantity of alcohol. It is possible, however, that prolonged use of librium might have led to some noticeable effect. It is important that patients who have to use sedatives, anti-tension compounds, and tranquilizers beware of losing control of themselves by drinking, for, although these drugs might not potentiate alcohol, they may well have an additive effect.

588. Holten, C. H., and Larsen, V.

THE POTENTIATING EFFECT OF BENACTYZINE DERIVATIVES AND SOME OTHER COMPOUNDS ON EVIPAL ANAESTHESIA IN MICE.

Acta Pharmacol. (Copenhagen), 12: 346-363 (48 ref.), 1956.

E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – nerv. syst. – anesthetics – *CAAAL-0 A-1404.

A series of compounds related to benactyzine, adiphenine, diphenhydramine, and also some commonly-used drugs, including local anesthetics and tranquilizers, were tested for their anesthesia-prolonging effects and spasmolytic properties. Aqueous solutions of test agents were injected ip into male mice. Most animals were anesthetized with 100 mg/kg evipal ip 30 min after pretreatment. In 1 test,

ethylallylbarbituric acid and ethanol were substituted for evipal, and their effects on sleeping time after benactyzine pretreatment were determined. Effects on sleeping times, and the spasmolytic activity of guinea pig jejunum in vitro were noted in some experiments, and LD₅₀ values were determined in acute toxicity tests. When ethanol (4 ml/kg, injected ip 30 min after 10 mg/kg benactyzine ip) was used as anesthetic, pretreated mice went more rapidly into anesthesia than did non-pretreated controls—5 min from injection to full anesthesia, as compared to 9 min for controls. Mean sleeping time was prolonged from the control value of 9 min to 16.5 min.

589. Hopkins, H. H.

SNAKE-BITE AND ALCOHOL.

Medical Record (New York), 36: 431 (0 ref.),

1889.

E – general – case hist. – DC (antidotal) – humans – *CAAAL-0

A-0790.

A case is given of a child bitten 3 times on the leg by a copperhead snake. The mother gave the child 1/2 pint of common whiskey just after it was bitten. The author found the leg much swollen and very painful. He applied hot water cloths to the limb, but gave no medicine. In 4 days, the swelling had almost disappeared, and the child had resumed normal activity. The author states that, in his experience, snake-bite always responds to moderate stimulation (alcohol or ammonia), and the quantity and quality of the whiskey given is generally responsible for the alarming symptoms occasionally met with, rather than the venom itself.

590. Hopkins, W. K.

ALCOHOL AS A REMEDY FOR THE POISON OF THE RATTLESNAKE.

Northwestern Medical and Surgical Journal (Chicago), 9(9): 389-391 (0 ref.),

1852-53.

E – general – case hist. – DC (antidotal) – humans – cardiovasc. – G.I. tract. – senses – *CAAAL-0

A-0791.

2 cases are presented, the first involving an adult male bitten by a snake above the ankle. The patient was given a 1/2 wine-glass of brandy, repeated in 5 min, and followed by a full glass 10 and 30 min later, for a total of 1 pint in 4 hr. The brandy cleared his vision, which had been impaired, restored his energy, and eliminated pain; by the next day, he was much improved. The second case concerns a 10-11 yr-old girl who was given brandy by the tablespoonful every 10 min after the bite until improvement was effected. The author is of the opinion that all the symptoms attending the bite of the rattlesnake can be accounted for by its prostrating effects on the nervous system; if the "sensorial power" of the victim is maintained, the consequences of the venom can be overcome.

591. Horn, G.

DRUGS AND ROAD TRAFFIC SAFETY.

Drugs Made in Germany (Aulendorf), 7: 20-22 (8 ref.),

1964.

E – exp. – general – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – med.-leg. – mot. vehic. – humans – mammals – blood lev. – other drug lev. – psychol. perform. – mot. perform. – CNS – anesthetics – barbiturates – tranquilizers – *CAAAL-0

A-0792.

The author reviews recent German experimental studies pertaining to drug use and driving, including studies on alcohol interaction with sedatives, anesthetics, antihistamines, and stimulants. Also discussed is the problem of basing medico-legal opinions on drug tests, in order to assess culpability for incompetence resulting from drug ingestion.

592. Horváth, D.

MENNYIBEN ÉS MILYEN FORMÁBAN BEFOLYÁSOLJÁK AZ ALKOHOLHATÁST

EGYES ÚGYNEVEZETT „DIVATOS” GYÓGYSZEREK. [To what extent and in what form

do some so-called "fashionable" drugs affect the action of alcohol?].

Orv. Hetil. (Budapest), 104: 2233-2236 (35 ref.),

1963.

H – general – review – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – CNS – amphetamines – stimulants – tranquilizers – *CAAAL-0 A-0793.

This review and general discussion covers the effects of the so-called "fashionable" drugs on alcohol. These drugs are largely outside the control of the physician. Of considerable importance are the minor tranquilizers, such as meprobamate and trioxazine, which potentiate the depressant effects of alcohol on the faculties of judgement and awareness. Stimulants such as aktedron and the antidepressant, ritalin, can impair driving ability, and, in combination with alcohol, can be especially dangerous; ritalin, when followed by alcohol, has the paradoxical effect of causing a leaden fatigue. One cannot readily predict the action of the drugs when combined with alcohol, and further study is warranted, as their use by the public is steadily increasing.

593. Hoskovec, J., and Štikar, J.

FARMAKA A ZPUSOBILOST K ŘÍZENÍ AUTOMOBILU. [drugs and driving ability].

Cesk. Psychiat. (Prague), 63(3): 195-198 (29 ref.),

1967.

C – SEC – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – barbiturates – tranquilizers – *CAAAL-0 B-0325.

The author reviews the problem of the effects of drugs on driving ability, with reference to the following groups: stimulants, psychomimetics, sedatives, barbiturates, antiprobics (e.g. meprobamate), tranquilizers (ataractics), and antihistamines. The individual effect and the type of accident to which each drug contributes are described. The combination of drugs and alcohol is mentioned—the effect may be potentiative, and may lead to a completely new syndrome of accident proneness. In Germany in 1961, 27% of the traffic accidents involved alcohol, and 5% involved both alcohol and drugs.

594. Höweler, A.

ALCOHOL ALS ANTIDOTUM BIJ ACUTE COCAINEVERGIFTIGING. [Alcohol as antidote in acute cocaine poisoning].

Nederl. T. Geneesk. (Amsterdam), 91: 2188-2192 (4 ref.),

1947.

D – ES – FS – GS – general – case hist. – DC (antidotal) – humans – anesthetics – *CAAAL-4787-V35 A-0794.

Alcohol po is recommended as an antidote in cocaine poisoning. The use of sedatives, such as veronal, to control spasms is strictly contraindicated, owing to the danger of paralysis. A case is reported of a female patient who was immediately heavily intoxicated by the submucous injection of 1/2 g cocaine into the nose. She recovered after receiving several sips of wine.

595. Hubach, H.

VERÄNDERUNGEN DER KRAMPFERREGBARKEIT UNTER EINWIRKUNG VON MEDIKAMENTEN UND WÄHREND DER ENTZIEHUNG. [Changes in spasmodic irritability through the effects of drugs, and during withdrawal].

Fortschr. Neurol. Psychiat. (Stuttgart), 31(4): 177-201 (278 ref.),

1963.

G – SEC – general – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – CNS – nerv. syst. – skel., muscle, skin – anesthetics – anticonvulsants – antispasmodics – barbiturates – sed., hypnot. – stimulants – *CAAAL-0 A-1452.

The withdrawal symptoms of various drugs are described, with particular reference to spasmodic irritability. It is stated that, despite the structural similarity of alcohol and paraldehyde, the former is classified as an anticonvulsive agent, and the latter as an antiepileptic agent. Reference is made to

the combined use of alcohol and paraldehyde (20 g paraldehyde in 60 g rum) in the treatment of neurasthenia. Because of the similar nature of withdrawal symptoms, the possible use of alcohol to combat the paraldehyde withdrawal syndrome, and vice versa, is suggested. Alcohol can be combined with a bromide, to be used as an antispasmodic, or with ether (2 parts ether: 1 part alcohol) for use as an inhalation mixture for narcosis. The arousal effect of picrotoxin, following administration of barbiturates, is reported to be decreased when the barbiturates are combined with alcohol. The author reclassifies alcohol as a hypnotic, narcotic, and antiepileptic compound, and its withdrawal syndrome is further discussed.

596. Hughes, D. T. D., Cramer, F., and Knight, G. J.
 USE OF A RACING CAR SIMULATOR FOR MEDICAL RESEARCH: THE EFFECTS OF
 MARZINE AND ALCOHOL ON DRIVING PERFORMANCE.
 Med. Sci. Law (London), 7: 200-204 (7 ref.), 1967
 E – exp. cont. – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – blood lev. –
 gastrointest. agents – *CAAAL-0 B-0326.

2 experiments were conducted on human subjects. The first tested the effects of marzine (an antihistamine sea-sickness remedy) alone on 5 subjects. In the second test, 2 subjects drove a racing car simulator under the following conditions: after no drug, after alcohol (100 ml of whiskey drunk over a 1/2 hr period, 3/4 hr prior to test—blood alcohol level less than 80 mg/100 ml), after marzine (50 mg taken 1 hr before test), and after marzine plus alcohol. Recorded data included min of racing time to cover 20 laps of the simulator circuit, number of mistakes, and racing time corrected for mistakes. The subjects did not differ significantly in performance. The performance of 1 subject, computed in corrected times, was: no drug—9.26 and 9.41 min, marzine—9.64 min, whiskey—11.85 min, and marzine plus whiskey—10.36 min.

597. Hughes, F. W., and Rountree, C. B.
 INFLUENCE OF ALCOHOL-TRANQUILIZER COMBINATIONS ON
 CHOICE-DISCRIMINATION IN RATS.
 Arch. Int. Pharmacodyn. (Gand), 133(3-4): 418-432 (7 ref.), 1961.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in
 vivo – blood lev. – psychol. perform. – CNS – barbiturates – tranquilizers – *CAAAL-9944-J2
 A-0795.

Rats were trained to avoid a shock stimulus. Anxiety reduction and choice discrimination were measured in groups of rats receiving alcohol (in doses of 0.5, 1.0 or 2.0 g/kg), selected tranquilizers (reserpine, 0.1 mg/kg; hydroxyzine, 5 mg/kg; chlorpromazine, 1.0 mg/kg; meprobamate, 50 mg/kg; or pentobarbital, 5 mg/kg), or combinations of both. It was concluded that, in small doses, alcohol plus pentobarbital effects a generalized depression in behaviour, resulting from additive depressant actions of each; the effect does not resemble tranquilization as operationally defined. Small doses of alcohol combined with reserpine, meprobamate, or hydroxyzine result in a greatly enhanced decrease of performance. The alcohol-chlorpromazine combination does not effect as great a decrease in performance. Both alcohol and the alcohol-tranquilizer combinations failed to lower anxiety.

598. Hughes, F. W., and Forney, R. B.
 ALCOHOL AND CAFFEINE IN CHOICE-DISCRIMINATION TESTS IN RATS.
 Proc. Soc. Exp. Biol. Med. (New York), 108: 157-159 (5 ref.), 1961.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in
 vivo – dose resp. – blood lev. – psychol. perform. – stimulants – *CAAAL-9997-J2 A-0796.

Rats received alcohol (1 g/kg), caffeine (50 mg/kg, 100 mg/kg, or 150 mg/kg), or alcohol-caffeine combinations, (using the same dosages), in a choice-discrimination test situation. By all measure-

ments, as well as observation, caffeine potentiated the depressive effect of alcohol long after the alcohol had disappeared from the blood. This was most marked with intermediate or with high dosages of caffeine. In no instance did caffeine antagonize the effects of alcohol.

599. Hughes, F. W., and Forney, R. B.
 DELAYED AUDIOFEEDBACK (DAF) FOR INDUCTION OF ANXIETY: EFFECT OF
 NORTRIPTYLINE, ETHANOL, OR NORTRIPTYLINE-ETHANOL COMBINATIONS ON
 PERFORMANCE WITH DAF.
 J.A.M.A. (Chicago), 185(7): 556-558 (9 ref.), 1963.
 E – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – dose resp. – blood lev. – psychol.
 perform. – senses – analg., antipyret. – antidepressants – *CAAAL-0 A-0797.

By means of a delayed auditory feedback system, an anxiety situation was created in 16 students. Alcohol in low dosage (blood level 50 mg/100 cc) effected a pronounced deficiency in performance. Nortriptyline (10 or 25 mg, 4 times/day po) did not alter performance. Neither the 10 nor the 25 mg doses of nortriptyline significantly enhanced or antagonized the action of ethanol, although a general tendency towards antagonism of ethanol was apparent. None of the combined doses produced untoward reactions greater than those produced by either drug alone.

600. Hughes, F. W., and Forney, R. B.
*STUDIES IN ANIMALS OF PHARMACOLOGIC ACTIONS RESULTING FROM
 COMBINATIONS OF ALCOHOL WITH OTHER CENTRALLY ACTING DRUGS.*
 Third International Meeting in Forensic Immunology, Medicine, Pathology, and Toxicology. Plenary
 Session II: "Drugs and driving." London, England, 7 pp. (1 ref.), 1963.
 E – exp. comp. – presentation – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals
 – acute admin. – in vivo – blood lev. – psychol. perform. – species or sex diff. – CNS – barbiturates
 – tranquilizers – *CAAAL-0 A-0798.

The experimental techniques developed in studying the potentiation of alcohol by other CNS depressants, and the calculation of a comparative tranquilizer index of psychotherapeutic drugs, are described. The over-all results of observations on rats and dogs, with respect to combinations of alcohol with the series of centrally-acting drugs, showed that potentiation occurred in the following (diminishing) order: reserpine, chlorpromazine, meprobamate, chlordiazepoxide, hydroxyzine, phenaglycodol, morphine sulphate, d-propoxyphene, codeine, pentobarbital.

601. Hughes, F. W., Forney, R. B., and Gates, P. W.
 PERFORMANCE IN HUMAN SUBJECTS UNDER DELAYED AUDITORY FEEDBACK
 AFTER ALCOHOL, A TRANQUILIZER (BENZQUINAMIDE) OR
 BENZQUINAMIDE-ALCOHOL COMBINATION.
 J. Psychol. (Provincetown), 55: 25-32 (15 ref.), 1963.
 E – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – psychol. perform.
 – CNS – senses – tranquilizers – *CAAAL-10081-J1 A-0799.

The verbal performance of 16 human subjects under the stimulus of delayed auditory feedback was evaluated after benzquinamide (25 mg po), alcohol (45 ml 200 proof alcohol/150 lb), or benzquinamide-alcohol. The results showed that benzquinamide increased performance, and alcohol effected a deficiency in performance. There was no evident synergism of the drug in combination with alcohol.

602. Hughes, F. W., Rountree, C. B., and Forney, R. B.
 SUPPRESSION OF LEARNED AVOIDANCE AND DISCRIMINATIVE RESPONSES IN
 THE RAT BY CHLORDIAZEPOXIDE (LIBRIUM) AND

ETHANOL-CHLORDIAZEPOXIDE COMBINATIONS.

J. Genet. Psychol. (Provincetown), 103: 139-145 (7 ref.), 1963.
 E – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – blood lev. – psychol. perform. – tranquilizers – *CAAAL-10625-J2 A-0800.

Chlordiazepoxide (10, 20, or 30 mg/kg ip) produced suppression of learned avoidance responses in rats, with little change in discriminative responses. Ethanol (0.5 mg/kg ip) caused reduction in avoidance responsiveness only when a diminution in performance (discrimination) resulted. The administration of alcohol in small amounts to chlordiazepoxide-treated rats caused a reversal of the avoidance-reducing property of this tranquilizer. This effect of alcohol on the drug persisted long after the alcohol had disappeared from the blood.

603. Hughes, F. W., and Forney, R. B.

DEXTRO-AMPHETAMINE, ETHANOL AND DEXTRO-AMPHETAMINE-ETHANOL COMBINATIONS ON PERFORMANCE OF HUMAN SUBJECTS STRESSED WITH DELAYED AUDITORY FEEDBACK (DAF).

Psychopharmacologia (Berlin), 6: 234-238 (8 ref.), 1964.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – other drug lev. – psychol. perform. – CNS – senses – amphetamines – *CAAAL-0 A-0801.

Mental tests were given to 8 human subjects in a delayed audiofeedback stress situation, and their performance quantified under the influence of d-amphetamine (20 mg) and/or ethanol (45 ml/150 lb). Ethanol decreased performance. D-amphetamine had relatively no effect, and, when given in combination with ethanol, no clear evidence of antagonism of ethanol by d-amphetamine was demonstrable, in fact, in the simple addition test, a synergistic effect was noted.

604. Hughes, F. W., and Forney, R. B.

COMPARATIVE EFFECT OF THREE ANTIHISTAMINICS AND ETHANOL ON MENTAL AND MOTOR PERFORMANCE.

Clin. Pharmacol. Ther. (St. Louis), 5(4): 414-421 (9 ref.), 1964.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – senses – autocoids – *CAAAL-0 A-0802.

Alcohol (50 ml/150 lb) impaired both the mental and motor performance in 16 paid volunteers; the three antihistaminics—clemizole (40 mg), diphenhydramine (50 mg), and tripeleennamine (50 mg)—did not. The effect of ethanol was not potentiated by the antihistaminics, but the action of diphenhydramine was potentiated by ethanol. The depressant properties of the antihistaminic drugs were much more apparent to the subjects than were those of alcohol, and yet, significant impairment was observed only with alcohol.

605. Hughes, F. W., Forney, R. B., and Richards, A. B.

COMPARATIVE EFFECT IN HUMAN SUBJECTS OF CHLORDIAZEPOXIDE, DIAZEPAM, AND PLACEBO ON MENTAL AND PHYSICAL PERFORMANCE.

Clin. Pharmacol. Ther. (St. Louis), 6(2): 139-145 (9 ref.), 1965.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – tranquilizers – *CAAAL-11185-J1 B-0085.

The effects of chlordiazepoxide (15 mg/day) and diazepam (6 mg/day), alone and in combination with ethanol (45 ml/150 lb), were studied in 18 human subjects. The drugs were administered 3

times/day for 2 days prior to the test, and once on the day of the test. Attentive motor performance was measured with a pursuit meter. Only ethanol was found to impair motor performance; over-all drug-ethanol interaction was not apparent, although, in 1 pattern, a synergistic effect of chlordiazepoxide with ethanol occurred. In mental performance, measured by a delayed auditory feedback system, ethanol alone effected a decrease. Drug-ethanol synergism was significant in the case of diazepam in 2 tests, but not in the case of chlordiazepoxide. With respect to the severity of symptoms, chlordiazepoxide and diazepam did not potentiate the subjective effects of ethanol, but, in fact, appear to have antagonized them.

606. Hugon, L.
UNE INTOXICATION PAR *COPRINUS ATRAMENTARIUS*. [Intoxication by *Coprinus atramentarius*].
Revue de Mycologie (Paris), 3, Suppl.: 48-49 (0 ref.), 1938.
F – general – DC (sensit.) – mot. perform. – cardiovasc. – G.I. tract – nerv. syst. – respir. – senses – miscellaneous – *CAAAL-0 A-1285.

A case is described in which the author and 7 other persons ate cooked *Coprinus atramentarius*. The people, including the author, who also drank wine were immediately ill, and the symptoms returned whenever wine was drunk during the following 48 hr. The effects noted were: difficulty in breathing, fast pulse, sensations in the limbs, hypnotic sleep afterwards, redness of the face, vomiting, exhaustion, difficulty in speech, loss of circulation in the toes, and a sensation of swelling of the feet. The next day, no wine was drunk and no sickness was felt. Neither bromide nor plant charcoal worked as remedies; oil of camphor and activated charcoal had some effect, but could not dispel the exhaustion. 1 person was confined to bed for 2 days. Persons drinking diluted wine suffered very little, and those who drank water experienced no ill effects at all. The author concludes that alcohol seems to enable the poison to be absorbed in the body, and that the intensity of the sickness also depends on individual gastro-intestinal sensitivity.

607. Hulpieu, H. R., Cole, V. V., and Smolenski, U.
THE FAILURE OF PYRUVATE AND ARSENITE TO ALTER ALCOHOL METABOLISM.
Quart. J. Stud. Alcohol (New Haven), 8(4): 553-568 (23 ref.), 1948.
E – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – cardiovasc. – liver, kidney – metab. proc. – miscellaneous – *CAAAL-3912-A2 A-0803.

The rate of decline of alcohol concentration in the blood, both with and without pyruvate administration, was studied in dogs. Ethanol was given at the rate of 1 g/kg iv. Pyruvate (3 times 5 g iv) had no effect on the rate of decline. The effect of sodium arsenite (5 mg/kg as 1% sol iv) was studied in rabbits and dogs. Arsenite did not retard the metabolism of alcohol, even in amounts approaching lethal doses.

608. Hulpieu, H. R., Forney, R. B., and Onyett, H. P.
THE FAILURE OF SUCCINATE TO ALTER THE METABOLISM OF ETHYL ALCOHOL IN DOGS AND RABBITS.
Quart. J. Stud. Alcohol (New Haven), 15: 9-15 (20 ref.), 1954.
E – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – species or sex diff. – absorp., distrib., stor. – *CAAAL-6693-A2 A-0804.

Rabbits were given 1 g alcohol/kg, followed by 250 mg succinate/kg iv, and dogs received the same alcohol dose plus either 250 mg succinate/animal or 20 mg/kg succinate. The succinate (administered in single, repeated, or continuous injections) failed to change the rate of disappearance of alcohol from the blood in the first stage of alcohol metabolism. No effect on the second stage of alcohol metabolism,

following succinate injections, could be demonstrated by determinations of the blood concentrations of acetaldehyde, pyruvic acid, or glucose.

609. Hunsicker, H.

ÜBER DIE AUFHEBUNG DER ALKOHOLISCHEN LEISTUNGSSCHÄDIGUNG DURCH PERVITIN. [The nullification by pervitin of the performance impairment caused by alcohol].

Dissertation, Medical Faculty of the University of Heidelberg, Germany, 15 pp. (10 ref.), 1941.
G – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – mot. perform. – CNS – amphetamines – *CAAAL-0 A-0805.

Experiments were conducted to study the effect of pervitin (15 mg sc) or pervitin in combination with alcohol (350 cc 40% brandy) in 1 subject given a running test. It was found that pervitin increased the tolerance limit to alcohol, and largely restored the performance impaired by alcohol to near normal. The subject finished a 3,000 meter running test (4 runs/condition) in the following times: under normal conditions—11 min 58 sec to 12 min 6 sec; after alcohol—13 min 29 sec to 14 min 2 sec; after alcohol plus pervitin—12 min 0 sec to 12 min 10 sec. No adverse side reactions were noted. The author concludes that pervitin eliminates inhibitory body mechanisms which prevent complete physical exhaustion, and prompts the body to use all its reserves. This can be very dangerous.

610. Hunt, R.

AN EXAMINATION OF THE TOXICITY OF ONE HUNDRED SAMPLES OF ILLICIT LIQUOR.

New Eng. J. Med. (Boston), 198(5): 230-234 (4 ref.), 1928.
E – exp. cont. – exp. comp. – congen. stud. – mammals – acute admin. – chronic admin. – in vivo – cardiovasc. – senses – *CAAAL-422-V1 A-1286.

The toxicities of 100 samples of illicit liquor were tested using mice, albino rats, and cats. The fatal and the tolerated doses, at a specified rate of injection, were determined in rats for each sample, and compared with the effects of dilutions of pure ethyl alcohol (EA) of the same strength. A popular “blended whiskey” was found to have the same toxicity as pure EA, whereas genuine bottled-in-bond (medicinal) whiskey was slightly more toxic. In both acute and chronic experiments, solutions in which up to 10% of the EA had been replaced by methyl alcohol were better tolerated than solutions containing EA only. Of the 100 samples of illicit liquor, 4 were found to be slightly more toxic than a 40 g/100 cc sol of pure EA; 3 of these had a slightly greater amount of EA. It is concluded that the toxicity of the illicit liquor tested closely paralleled its content of EA, and that there is no indication of substances significantly more poisonous than EA.

611. Hurst, H.

PRINCIPLES OF INSECTICIDAL ACTION AS A GUIDE TO DRUG REACTIVITY-PHASE DISTRIBUTION RELATIONSHIPS.

Faraday Society, Transactions (London), 39: 390-411 (46 ref.), 1943.
E – SEC – exp. comp. – DC (add., infra-add., unspec. incr.) – other org. – in vivo – absorp., distrib., stor. – alcohols – *CAAAL-0 A-0806.

Experiments were carried out, showing the different toxic interaction effects of various drugs on insect larvae. The survival time of *Calliphara erythrocephala* larvae in ethyl alcohol-octyl alcohol mixtures was shown to be shorter than in the pure drug components. The same effect was obtained with ethanol-kerosene on blowfly larvae. The opposite, or antagonistic, effect was demonstrated with cetyl alcohol, due to its strong capillary action retarding the biological activity of amyl alcohol.

612. Hurst, P. M., Radlow, R., Chubb, N. C., and Bagley, S. K.
EFFECTS OF ALCOHOL AND D-AMPHETAMINE UPON MOOD AND VOLITION.
Psychol. Rep. (Missoula), 24: 975-987 (14 ref.), 1969.
E – exp. cont. – exp. comp. – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – humans
– acute admin. – in vivo – psychol. perform. – amphetamines – *CAAAL-0 B-0525.

The effects of alcohol (A), d-amphetamine (D), and placebo, separately and in combination, on mood self-ratings and volitional behaviour related to changes in mood (gambling, risk-taking, verbal production) were measured in 70 male subjects acting as their own control. A (45 g/70 kg) increased risk-taking, but not verbal production; D (14 mg/70 kg) increased verbal production, but not risk-taking. The combination of A + D was not resolvable from the separate effects of the components. Approximately additive effects were seen with “urgency”, “social affection”, “egotism”, “elation”, and “confidence”, all of which are generally suggestive of euphoria. In “concentration”, however, the results of A + D were closely comparable to those of A alone. A sub-additive effect was evident with “vigour”, and the A + D effect was comparable to either A or D separately. Supra-addition was suggested in “fatigue” and “anxiety”, the A + D combination yielding a “fatigue” reduction exceeding the simple addition of effects by 24%, and the “anxiety” reduction exceeding simple addition by 74%.

613. Husemann, T.
ANTAGONISTISCHE UND ANTIDOTARISCHE STUDIEN. [Antagonistic and antidotal studies].
Naunyn Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 10: 101-124 (17 ref.), 1879.
G – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – gastrointest. agents – stimulants – *CAAAL-0 A-0807.

A detailed analysis of the alleged antagonism of alcohol and strychnine was made in a series of tests on rabbits. In 3 experiments, sc injections of 4.0, 5.0, and 5.5 g 50% alcohol in water were administered. 1 hr 20 min later, 0.7 or 0.8 mg sc strychnine dissolved in water and hydrochloric acid, followed by 1 g 50% alcohol sc, was given. All animals recovered. In a second series, 1 rabbit received 10 g 50% alcohol, and, after 40 min, 1.2 mg strychnine and 2 g 50% alcohol; the animal died after 25 min. A second rabbit was given 14 g 50% alcohol, and, after 35 min, 2.2 mg strychnine plus 2 g 50% alcohol; it died after 3 hr. The author concludes that the established lethal doses of strychnine cannot be influenced by alcohol, and that alcohol intoxication is not altered by strychnine.

614. Hutchens, J. O., Wagner, H., Podolsky, B., and McMahon, T. M.
THE EFFECT OF ETHANOL AND VARIOUS METABOLITES ON FLUOROACETATE POISONING.
J. Pharmacol. Exp. Ther. (Baltimore), 95: 62-70 (10 ref.), 1949.
E – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev.
– species or sex diff. – CNS – metab. proc. – respir. – *CAAAL-0 A-0808.

Protection against fluoroacetate poisoning by ethanol was investigated in mice, guinea pigs, rabbits, and dogs. Mortality among fluoroacetate-poisoned mice, guinea pigs and rabbits was significantly reduced when about 800 mg/kg of ethanol was administered sc as a 10% ethanol sol in saline within 30 min of poisoning. The most striking effects were obtained in mice when the ethanol was given within 10 min of poisoning. Ethanol counteracted twice the LD₅₀ of fluoroacetate in mice, and saved significant numbers of guinea pigs and rabbits from fluoroacetate doses barely greater than the LD₅₀, but was of little benefit in dogs. The effect of other substances and the mechanism of protection of ethanol is discussed.

615. Iaroshevski, S.
STRIKHNIN KAK ANTAGONIST ALKOGOLIA. [Strychnine as an antagonist of alcohol].
 Meditsinskoe Obozrenie (Moscow), 27: 332-341 (5 ref.), 1887.
 R – exp. cont. – DC (decrease) – mammals – chronic admin. – in vivo – CNS – stimulants –
 *CAAAL-0 A-1334.

To study the effect of strychnine as an antagonist of alcohol, the author performed a series of experiments on 6 dogs divided into 2 groups. The first group received only chronic alcohol intake, and 1 of the dogs died within 30 days from the beginning of the experiments, after receiving a total of 45 oz of 65% alcohol. The other 2 animals in the group died within 7 days after receiving a total of 11 and 20 drams of 42% alcohol, respectively. The second group received chronic administration of alcohol in conjunction with strychnine sol (1 grain/oz water concentration), and 1 animal died within 28 days, after receiving 146 drams of 42% alcohol plus 140 drops of strychnine. The remaining 2 dogs received 146 drams of 42% alcohol plus 140 drops of strychnine in 29 days, and 756 oz of alcohol (374 oz of 65% and 382 oz of 42%) plus 19.8 g of strychnine within 109 days, respectively. They both survived the experiment, but were killed for dissection. From the dissection findings, the author concludes that strychnine is an excellent antagonist of alcohol. Strychnine allows the organism to withstand chronic administration of large amounts of alcohol for long periods of time without any significant damage or intoxication. The author advocates the therapeutic use of strychnine for all forms of alcoholism, despite the fact that, beyond a certain limit in dosage, the effect of strychnine is highly poisonous.

616. Iaroshevski, S.
ESHCHE O STRIKHNIN, KAK ANTAGONIST ALKOGOLIA. [More about strychnine as an antagonist of alcohol].
 Meditsinskoe Obozrenie (Moscow), 29:194-201 (O ref.), 1888.
 R – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – CNS – liver, kidney – stimulants –
 *CAAAL-0 A-1287.

In a sequel to an earlier article (Meditsinskoe Obozrenie, 27: 332-341, 1887), an experiment is reported concerning the effect of acute administration of alcohol and strychnine on 2-16 lb dogs. The first dog received 3 1/2 oz of 62% alcohol in a period of 10 min, and the other received 9 1/2 oz of 62% alcohol + 8 injections of strychnine sol (1 grain/oz water) within 20 min. Signs of intoxication appeared in the first case after the first oz of alcohol. The other animal behaved quite normally, even after 5 oz of alcohol, and lived for 2 1/2 hours after the test. The author then studied the dissected organs, and compared the findings. It is concluded that strychnine effectively counteracts the intoxicating effect of alcohol, even when the latter is administered acutely. Furthermore, strychnine protects the liver, kidneys, and the CNS from damage. The author suggests extensive therapeutic use of strychnine, especially in nervous forms of alcoholism.

617. Ideström, C. -M.
FLICKER-FUSION IN CHRONIC BARBITURATE USAGE: A QUANTITATIVE STUDY IN THE PATHOPHYSIOLOGY OF DRUG ADDICTION.
 Acta Psychiatrica et Neurologica Scandinavica (Copenhagen), Suppl. 91: 93 pp. (36 ref.), 1954.
 E – exp. cont. – exp. comp. – cross-tol. – humans – acute admin. – chronic admin. – in vivo – blood lev. – analg., antipyret. – barbiturates – *CAAAL-7103-D1 A-0809.

Flicker-fusion proved to be the most sensitive and reliable test for the measurement of the effects of various barbiturates. Tolerance tests were carried out with oral administration of 0.25-0.30 g/70 kg phenobarbital/day for from 2-13 months in 4 epileptic subjects; in 1 case, 150 ml 40% alcohol, taken orally on the 297th day, was slightly stimulating (blood alcohol—0.06°/oo, thus indicating that cross-tolerance may exist between barbiturates and alcohol. In 9 cases of chronic barbiturate intoxication, alcohol (40-175 ml) was given before and after withdrawal of drug (blood

alcohol—0.41-1.43°/oo). It was again demonstrated that cross-tolerance between barbiturates and alcohol exists, and that tolerance doses of alcohol in cases of barbiturate addiction produce a stimulating effect. In addition, patients receiving 0.25-1.00 g dihydrocodeinone exhibited a cross-tolerance for alcohol, and, after withdrawal, alcohol caused a strong depressive effect.

618. Iida, S., and Yuguchi, T.

ARUKÖRU TAISHA NO RATE-LIMITING FACTOR NI TSUITE. [the rate-limiting factor in alcohol metabolism].

Folia Pharmacol. Jap. (Nippon Yakurigaku Zasshi) (Kyoto), 57: 577-584 (32 ref.), 1961.
J – exp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – liver, kidney – metab. proc. – elect., water-bal. agents – nutritive agents – *CAAAL-10227-A2

A-0810.

Administration of pyruvate and fructose accelerated the rate of alcohol metabolism in the rabbit only when the initial rate of alcohol metabolism was less than maximal. An increase in the diphosphopyridine nucleotide concentration in the liver, produced by administration of nicotinamide, failed to increase alcohol metabolism. The rate-limiting factor is apparently alcohol dehydrogenase, but there are other factors which control the rate of alcohol oxidation, “particularly in fasted and carbon tetrachloride-poisoned rabbits.”

619. Im Obersteg, J., and Bäumler, J.

UNFÄLLE UNTER DER EINWIRKUNG VON ARZNEIMITTELN UND ALKOHOL. [Accidents under the influence of drugs and alcohol].

Schweiz. Med. Wschr. (Basel), 97(32): 1039-1042 (10 ref.), 1967.

G – ES – SEC – stat. surv. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – blood lev. – other drug lev. – analg., antipyret. – barbiturates – sed., hypnot. – tranquilizers – *CAAAL-0 B-0327.

Of 328 patients hospitalized after traffic, occupational, and non-occupational accidents, the blood alcohol concentration was determined and the urine tested for drugs (mainly barbiturates, tranquilizers, sedatives, and stimulants). 19% (61) of the blood samples were alcohol positive. In 2/3 of the traffic accidents, the blood alcohol concentration was over 0.8°/oo. In more than 100 patients, drugs were found in the urine, but only for 4% (13) could it be definitely established that the drug had been taken prior to the accident, and not afterwards during emergency treatment. In this study, alcohol was a much more important factor in the accidents than were the drugs, but it was assumed that, in about 4% of the cases, drug interaction with alcohol was involved.

620. Imrie, J. A.

EMERGENCIES IN GENERAL PRACTICE: ACUTE ALCOHOLIC POISONING.

Brit. Med. J. (London), 2: 428-430 (1 ref.), 1955.

E – general – DC (antidotal) – DC (add., infra-add., unspec. incr.) – psychot. humans – drug-dep. humans – blood lev. – mot. perform. – absorp., distrib., stor. – cardiovasc. – CNS – respir. – analg., antipyret. – autonomic agents – diagnost. agents – nutritive agents – stimulants – *CAAAL-7346-N6 A-0811.

The progressive states of the excessive drinker's clinical condition are discussed, and the possible dangers emphasized. In cases of ethanol poisoning, the administration of morphine, morphine-scopolamine, or barbiturates can be fatal. Intoxication may be treated with nikethamide (5-10 ml, iv or im), caffeine (1 g iv), sodium benzoate (1 g iv), or vitamin B₆ (50-100 mg iv). More intensive vitamin therapy may consist of 1,000 mg vitamin B₁, 200 mg vitamin B₆, and 1,500 mg vitamin C—especially after prolonged bouts. Treatment of poisoning by the other alcohols is also discussed.

621. Isaac, M., Bovill, J. G., Dundee, J. W., and Pandit, S. K.
 CLINICAL STUDIES OF INDUCTION AGENTS. XXXV: STUDIES ON COMBINATION
 OF ETHANOL WITH METHOHEXITONE AND DIAZEPAM.
 Brit. J. Anaesth. (Altrincham), 42(6): 521-523 (3 ref.), 1970.
 E – FS – GS – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo
 – CNS – G.I. tract – liver, kidney – metab. proc. – respir. – skel., muscle, skin – anesthetics – autocoids
 – tranquilizers – *CAAAL-0 B-0526.

The use of ethanol in combination with methohexitone or diazepam as an iv anaesthetic was evaluated in 80 patients. 4 techniques of anaesthesia were employed: 1) atropine premedication—up to 350 ml ethanol (8% w/v sol), followed by 0.8 mg/kg methohexitone; anesthesia continued with nitrous oxide-oxygen, 2) same as above except that methohexitone preceded ethanol, 3) diazepam (10 mg), atropine (0.6 mg) premedication—up to 450 ml ethanol, and 20-30 mg diazepam iv when drowsy; anesthesia continued with nitrous oxide-oxygen, 4) same as for (3) except 10-15 mg diazepam. Methohexitone abolished ethanol induction delirium, but typical methohexitone side-effects occurred in 1/2 of the patients. Recovery was prompt, but with a marked incidence and severity of emergence delirium. No relationship was found between the order of acute administration and the degree of potentiation or synergism between alcohol and barbiturates. In the 2 diazepam-ethanol series, induction was uneventful, but recovery was markedly delayed. It is concluded that combinations of ethanol with methohexitone or diazepam are clinically unacceptable for induction of anaesthesia for minor surgery.

622. Israel, Y., Khanna, J. M., and Lin, R.
 EFFECT OF 2,4-DINITROPHENOL ON THE RATE OF ETHANOL ELIMINATION IN
 THE RAT IN VIVO.
 Biochem. J. (London), 120(2): 447-448 (13 ref.), 1970.
 E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – other drug lev. – absorp., distrib.,
 stor. – liver, kidney – metab. proc. – respir. – skel., muscle, skin – miscellaneous – *CAAAL-0
 B-0939.

Male Wistar rats (90-100 g) received 3.4 mg/100 g of 2,4-dinitrophenol ip, followed 30 min later by 112 or 304 mg/100 g ethanol ip. After complete homogenation, the amount of ethanol was determined enzymatically, and the rate of ethanol elimination calculated; the amount of ethanol expired or lost by cutaneous evaporation was also determined. In each of 20 pairs, the dinitrophenol-treated rat showed a 20-30% greater loss of ethanol. The rate of ethanol disappearance was 50% greater in dinitrophenol-treated rats if calculated on the difference between 60 and 120 min values for residual ethanol after the larger dose. The amount lost by expiration and evaporation was 2.7-8.2% of total ethanol eliminated in untreated rats. Dinitrophenol increased this rate, but only enough to account for 7-25% of the dinitrophenol effect, thus indicating a true metabolic effect. The results suggest that the rate-limiting step in the oxidation of ethanol is the reoxidation of NADH to NAD⁺, rather than alcohol dehydrogenase activity. This would explain the ability of pyruvate and fructose to increase the rate of ethanol metabolism in vivo.

623. Issekutz, B. von
 STUDIEN ÜBER METHÄMOGLOBINBILDUNG. XV. MITTEILUNG. [Studies on
 methemoglobin formation. XV].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 193:
 551-566 (7 ref.), 1939.
 G – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin.
 – in vivo – dose resp. – blood lev. – blood comp., sites, lymph – *CAAAL-0 A-1453.

Methemoglobin formation after the administration of methemoglobin-forming drugs, and the influence thereon of simultaneous alcohol administration, were investigated in cats (1700-3000 g). After

pretreatment with 0.5 g urethan sc, the following substances were administered sc, with or without simultaneous injection of 0.3 cc of a 10% sol of 96% alcohol sc: *p*-aminophenol (8-15 mg/kg); phenylhydroxylamine (0.5-2 mg/kg); *m*-, *o*-, and *p*-dinitrobenzene (0.12-0.25, 5-30, and 1-10 mg/kg, respectively); and sodium nitrite (10-30 mg/kg). Blood samples were taken at regular intervals and analyzed for methemoglobin. Methemoglobin formation by sodium nitrite, *o*-dinitrobenzene, or *p*-dinitrobenzene was unaffected by alcohol, whereas the alcohol caused a significant increase when administered with *p*-aminophenol, phenylhydroxylamine, and *m*-dinitrobenzene. In the case of the sodium nitrite-alcohol combination, it is concluded that the action of the former is too rapid for the latter to have any influence. No explanation is given for the effect of alcohol on the other substances, but the possibility of poisoning through a combination of alcohol and *m*-dinitrobenzene is noted.

624. Iwai, J.
SEKKAI CHISSO CHŪDOKU NI OKERU KETSUEKI NO HENKA TO *ARUKŌRU* TO NO KANKEI. [Changes in blood due to calcium cyanamide poisoning, and their relation to alcohol intake].
Fukuoka Acta Medica (Fukuoka-Ikwadaigaku-Zasshi) (Fukuoka), 25(2): 2208-2241 (33 ref.), 1932.
J – exp. cont. – DC (sensit.) – mammals – acute admin. – chronic admin. – in vivo – acid-base, blood pH, elect. – blood comp., sites, lymph – miscellaneous – *CAAAL-0 A-1288.
Due to the increasing possibility of alcohol-intolerance reactions occurring among workers involved with calcium cyanamide (CC), experiments were conducted to determine the influence of CC on the blood. Rabbits received large and small acute doses of CC by inhalation or po. The results showed no change in hemoglobin concentration, but there was an increase in hemolysin and agglutinin levels. The amboceptor concentrations in the hemolysins were higher, regardless of length of time between doses. 2 hr after acute CC poisoning, higher levels of calcium, protein nitrogen, non-protein nitrogen, and pH were found; when alcohol and CC were administered simultaneously, these levels approached those of the control group. Chronic CC poisoning produced no notable change in the production of amboceptors.
625. Iwai, J.
SEKKAI CHISSO CHŪDOKU NI OKERU KETSUEKI NO HENKA TO *ARUKŌRU* TO NO KANKEI (DAI NI HŌKOKU). [Changes in blood due to calcium cyanamide poisoning, and their relation to alcohol intake (second communication)].
Fukuoka Acta Medica (Fukuoka-Ikwadaigaku-Zasshi) (Fukuoka), 27(7): 1717-1728 (16 ref.), 1934.
J – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – mammals – acute admin. – in vivo – blood comp., sites, lymph – *CAAAL-0 A-1405.
Due to the occurrence of several cases of calcium cyanamide poisoning, especially after concomitant alcohol consumption, the influence exerted on blood by calcium cyanamide, alone and in combination with alcohol, was investigated in rabbits. 24 hr after administration of cyanamide or cyanamide plus alcohol, blood analyses were performed. It was found that cyanamide alone increased the number of pseudo-eosinophilic cells by up to 24%, while this increase was raised to 38% by cyanamide plus alcohol. After cyanamide alone, the red blood cells with reticular structure numbered 19-23 for every 500 normal red blood cells. After cyanamide plus alcohol, however, this number was only 12-17. With material which had been exposed to air for 2 months, no reticular erythrocytes were observed.
626. Jacobsen, E.
THE METABOLISM OF ETHYL ALCOHOL.
Pharmacol. Rev. (Baltimore), 4: 107-135 (153 ref.), 1952.
E – SEC – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – mammals

– acute admin. – in vivo – in vitro – blood lev. – absorp., distrib., stor. – liver, kidney – metab. proc.
 – indust. intox. – unclass. ther. agents – *CAAAL-6170-A3 A-0812.

In this general review, the author notes that, of all the drugs examined to date, only dinitrophenol and dinitrocresol have been found to accelerate alcohol metabolism, and, since very high and toxic doses of dinitrophenol are necessary to demonstrate the effect, it is impractical for human use. Phloridzin inhibits alcohol metabolism by about 25%, and cyanamide and antabuse, which strongly inhibit the second step of alcohol oxidation, also affect the first stage, though to a lesser degree. The practical application of these and other drugs to man is somewhat limited by the lack of reliable information.

627. Jaillet, J.

DE L'ALCOOL: SA COMBUSTION, SON ACTION PHYSIOLOGIQUE, SON ANTIDOTE.

[Alcohol: its combustion, its physiological action, its antidote].

Dissertation, Faculty of Medicine of the University of Paris, France, 178 pp. (5 ref.), 1884.

F – exp. – DC (decrease) – mammals – acute admin. – in vivo – blood comp., sites, lymph – cardiovasc.

– CNS – G.I. tract – respir. – stimulants – *CAAAL-0 A-0813.

The study was conducted in 3 parts: combustion of alcohol, physiological and toxic action of alcohol, and treatment of acute alcoholic poisoning by administration of strychnine. Experiments on dogs partially confirmed Amagat's conclusions, drawn from experiments on rabbits (Journal de Thérapeutique, 3: 378-383, 1876), that strychnine counteracts the effects of fatal doses of alcohol; however, the two substances are not true antagonists. The bulbar stimulation of strychnine counteracts the paralysis of the respiratory center produced by alcohol, and it is for this reason that death can be prevented. On the other hand, even toxic doses of alcohol cannot counteract the effects of strychnine poisoning. Large doses of strychnine can be safely used in cases of alcoholic coma or collapse. Linking his study to previous research on humans, the author considers a good treatment of acute alcoholic poisoning to consist of the administration of 1 cg of strychnine injected sc, followed by small doses every 2 hr, until reflexive movements show improvement.

628. Janiszewski, H.

„ERGÄNZENDE STELLUNGNAHME“ DES BUNDESGESUNDHEITSAMTES ZUM GUTACHTEN „ALKOHOL BEI VERKEHRSSTRAFTATEN“. [“Supplementary statement” of the Federal Department of Health regarding the expert's report on “Alcohol in traffic offences”].

Blutalkohol (Hamburg), 5(2): 112-119 (5 ref.), 1968.

G – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – barbiturates – sed., hypnot. – tranquilizers – *CAAAL-0 B-0328.

The author appends a supplementary statement to the report by the Health Ministry of the Federal Republic of Germany (see: Lundt, P. V. et al., eds.: *Alkohol bei Verkehrsstraftaten; ergänzende Stellungnahme zu den bisher vorgelegten Gutachten des Bundesgesundheitsamtes zur Frage*. [Alcohol in traffic offences; supplementary statement to the expert opinions of the Federal Department of Health on the question]. Bad Godesberg: Kirschbaum, 18-27, 1967). He attempts to answer 3 questions: 1) “How do disease, injuries, and drugs affect the alcohol metabolism in man?”; 2) “How reliable is the test tube procedure “alcotest?”; and 3) “What is the forensic value of clinical reports with reference to the determination of a driver's impairment?” The great difficulties of meaningful experiments, under realistic conditions, on the interaction of alcohol and other drugs are emphasized. An increase of alcohol oxidation through some drugs is established, but is not important in driving conditions. The interaction of alcohol and barbiturates can be dangerous and has to be pointed out more clearly. “Sobering-up” medications are not only ineffectual, but often dangerous.

629. Jansen, G.
 EXPERIMENTELLE UNTERSUCHUNGEN IN FORM VON PHARMAKOLOGISCHEN ARBEITSVERSUCHEN. [Experimental studies in the form of pharmacological tests during work].
 Med. Exp. (Basel), 2(2-4): 209-216 (4 ref.), 1960.
 G – ES – FS – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – metab. proc. – amphetamines – stimulants – *CAAAL-10239-J1 A-0814.

Human subjects were accustomed to the Graf driving device, and then subjected to distracting stimuli—which failed to affect performance. Alcohol, caffeine, pervitin, preludin, and somnifen affected performance; 1 g/kg alcohol greatly decreased the level of coordination and judgment. After 0.5 g/kg alcohol, administration of 9 mg pervitin offset the alcohol effect almost completely, but 0.29 g of caffeine failed to neutralize the alcohol impairment. 9 mg pervitin compensated for the action of 1 g/kg alcohol, up to a blood alcohol level of 0.6 mg%. The compensatory effect of pervitin applied only to coordination; it did not apply to other functions impaired by alcohol, such as equilibrium, judgment, and self-criticism. Neither pervitin nor caffeine influenced the oxidation rate of alcohol.

630. Janz, D.
 ANTIEPILEPTIKADOSIERUNG NACH ALKOHOLEXZESS. [Antiepileptic dosage after alcohol excess].
 Deutsch. Med. Wschr. (Stuttgart), 95: 651 (0 ref.), 1970.
 G – abst. – general – DC (unchanged) – humans – CNS – anticonvulsants – *CAAAL-0 B-0527.

A question is asked concerning an epileptic accustomed to a relatively high anticonvulsive dose of zentropil and mylepsin (1 tablet 3 times /day), who goes into a status epilepticus after alcoholic intoxication. If switched to other medication, can he receive parenterally the same dosages of standard anticonvulsants (valium, epanutin, luminal, or distraneurin), despite the high quantity of alcohol? The answer is given that, according to experience, epileptic attacks usually do not occur during alcoholic intoxication, but only afterwards, and a status epilepticus developing after alcohol excess can be treated according to the usual rules and in the usual dosage. Insofar as respiration and circulation of the blood are not considerably affected, the medication as indicated can be used without hesitation. A daily medication of 300 mg zentropil and 750 mg mylepsin does not appear too high, and should be adequate. No change in medication is therefore deemed necessary.

631. Jaulmes, C., Delga, J., and Gary Bobo, C.
 MÉTABOLISME DE L'ALCOOL CHEZ LES ANIMAUX SOUMIS À L'HIBERNATION ARTIFICIELLE. [Alcohol metabolism in animals under artificial hibernation].
 C.R. Soc. Biol. (Paris), 150: 1094-1097 (7 ref.), 1956.
 F – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – metab. proc. – analg., antipyret. – sed., hypnot. – tranquilizers – *CAAAL-8085-A2 A-0815.

Rabbits received 10 ml of a 20% alcohol sol/kg by stomach tube. When alcohol alone was administered, maximum blood alcohol concentration was reached 1-2 hr after administration. Animals put under artificial hibernation were given 2 injections, 30 min apart, of 0.5 ml/kg of a mixture containing 50 mg chlorpromazine, 50 mg promethazine, and 50 mg pethidine. Alcohol (above dose) was given immediately after the second injection of the mixture, and the animals were plunged into cold water; 1-2 additional injections of the mixture were then given. The blood alcohol curve was considerably altered—the peak was not reached until about the 7th hr, and this level was maintained until revival. 2 further variant experimental series followed. It is concluded that hypothermia inhibits the diffusion

of alcohol into the blood stream. Both the hypothermia and neuroplegic drug mixture retarded alcohol oxidation.

632. Jetter, W. W.

WHEN IS DEATH CAUSED BY OR CONTRIBUTED TO BY ACUTE ALCOHOLISM?

Clinics (Philadelphia), 1(6): 1487-1502 (7 ref.),

1943.

E – SEC – general – DC (add., infra-add., unspec. incr.) – post-mort. – blood lev. – other drug lev.

– G.I. tract – respir. – barbiturates – *CAAAL-3857-D1

A-0816.

Case histories were used to determine the causal relationship between alcohol and death. It was found that death can occur several hr, and even days, after the last alcohol ingestion. If death occurs more than 24 hr after the last ingestion, no alcohol will be found in the blood after death; the blood condition is nevertheless predetermined by irreversible changes caused by alcohol ingestion. If death occurs, and the deceased did not drink to excess, synergism between alcohol and other drugs such as barbiturates must be assumed and properly investigated. A case is described in which a 45 yr-old male died from the synergistic action of alcohol and phenobarbital. A post-mortem examination revealed a blood alcohol concentration of 0.23%; a large amount of phenobarbital was present in the gastrointestinal tract, and a small amount was present in the brain and blood.

633. Jetter, W. W., and McLean, R.

POISONING BY THE SYNERGISTIC EFFECT OF PHENOBARBITAL AND ETHYL ALCOHOL: AN EXPERIMENTAL STUDY.

Arch. Path. (Chicago), 36: 112-122 (6 ref.),

1943.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp.

– blood lev. – other drug lev. – CNS – skel., muscle, skin – barbiturates – *CAAAL-3921-D2

A-0817.

By testing 52 rats, a max sublethal dose of 8 mg/g was determined for ethanol, and 0.20 mg/g determined for phenobarbital. Max sublethal doses or fractions thereof were used in combination on 31 rats. The max sublethal dose of one drug plus half that of the other caused fatalities in a high percentage of animals. It is concluded that the synergistic action of the drug and alcohol depends on the fact that both are CNS depressants and exert a combined paralyzing effect on the respiratory center. 3 cases of accidental deaths of humans who ingested alcohol plus phenobarbital or pentobarbital indicate a similar fatal synergism in man—neither the drug nor the alcohol were taken in sufficient quantities to individually cause death.

634. Jofre de Breyer, I. J., and Soehring, K.

WIRKUNGEN VON AETHANOL AUF DEN ACETANILID-STOFFWECHSEL IN RATTENLEBERSCHNITTEN. [Effects of ethanol on the metabolism of acetanilide in rat liver slices].

Arzneimittelforschung (Aulendorf), 18(5): 604-605 (8 ref.),

1968.

G – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – in vitro – dose resp. – liver,

kidney – analg., antipyret. – *CAAAL-0

B-0329.

72 rat liver slices (introduced into a Ringer sol, exposed to 37°C under oxygen, and stabilized in air) were incubated with acetanilide (37μM) and varying ethanol concentrations (17μM, 17mM and 85 mM). More than 95% of the charged acetanilide was recovered (in the control, and with both of the low ethanol concentrations) as free and conjugated hydroxyacetanilide. At an ethanol concentration of 85 mM, the recovered percentage was considerably lower. The author concludes that high concentrations of ethanol inhibit hydroxylation.

635. Johnson, F. H., Eyring, H., and Kearns, W.
A QUANTITATIVE THEORY OF SYNERGISM AND ANTAGONISM AMONG DIVERSE INHIBITORS, WITH SPECIAL REFERENCE TO SULFANILAMIDE AND URETHANE.
 Arch. Biochem. (New York), 3(1): 1-31 (22 ref.), 1943.
 E – SEC – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – other org. – in vivo
 – dose resp. – anti-infectants – *CAAAL-0 A-0818.

It was hypothesized that sulfanilamide and urethane, which act directly on the bacterial luminescent system, also enter into a loose reversible combination with each other, and that accurate, quantitative predictions of the effects of a wide range of concentrations of both inhibitors mixed in various proportions can be made. Furthermore, it was predicted that, at various temperatures and concentrations, antagonism and synergism may be expected in the inhibition of luminescence by sulfanilamide plus ether, alcohols, chloroform, acetone, and certain other substances. Experimental data verified these predictions. It was found that lower concentrations of ethanol, in the presence of 0.003 M sulfanilamide at 5°C and 22°C, decreased the amount of sulfanilamide inhibition. Higher concentrations of ethanol, though still combining with sulfanilamide according to the same equilibrium constant as previously, gave rise to a greater inhibition than with sulfanilamide alone. The same result could be achieved by raising the temperature—at low temperatures the action of ethanol was antagonistic, and, at higher temperatures, synergistic.

636. Johnston, W. W.
ALCOHOLISM AND OPIUM EATING.
 Medical Society of the District of Columbia, Transactions (Washington), 2: 35-41 (0 ref.), 1875.
 E – SEC – general – case hist. – conj. addict. – post-mort. – drug-dep. humans – mot. perform. – psychol. perform. – cardiovasc. – liver, kidney – respir. – skel., muscle, skin – hallucinogens – *CAAAL-0 A-1289.

Case histories involving combined alcoholism and opium addiction are discussed. The main case presented is that of a man who commenced the constant use of morphine hypodermically to relieve the pain of rheumatism. Over the years, he indulged increasingly in morphine and alcohol, until, after 8 yr, he was taking 30 or more grains of morphine and about 1 quart of whiskey daily. When treated in hospital, his intake could not be reduced below 2 1/2 grains of morphine and 18 oz of whiskey daily. The morphine could be reduced, provided the whiskey administration was maintained. He died 9 yr after beginning the habit. An autopsy revealed the heart, kidneys, liver, and spleen to be very large and heavy, with extensive infiltration of fat. These lesions were attributed to the alcoholism, whereas the morphine had the opposite effect of producing a wasting of the tissues. Death was attributed to imperfect contraction of the heart from fatty degeneration.

637. Jones, R. T., and Stone, G. C.
PSYCHOLOGICAL STUDIES OF MARIJUANA AND ALCOHOL IN MAN.
 One hundred and twenty-fifth Annual Meeting of the American Psychiatric Association, Bal Harbour, Florida, 18 pp. (15 ref.), May, 1969.
 E – SEC – exp. cont. – cross-tol. – humans – acute admin. – chronic admin. – in vivo – mot. perform. – psychol. perform. – CNS – *CAAAL-0 B-0940.

10 regular male users of marihuana were given smoked and orally-administered marihuana, a placebo, or alcohol, and performance on digit symbol, rod and frame, and time estimation tests was rated. It was found that the subjects were unable to distinguish between smoked marihuana and placebo. The oral marihuana dose produced primarily dysphoric symptoms, and, in this respect, was similar to alcohol. The marihuana altered pulse rate, time estimation, and the EEG, but had no effect on the measure of field dependence or on the digit symbol task. Both marihuana and alcohol appeared to be mild intoxicants. The relative lack of behavioural change, and the general mild clinical picture after a dose of 1 cc 95% alcohol/kg (breathalyzer alcohol level 1 hr after ingestion—60-120 mg%)—almost

the equivalent of 4 shots of 100 proof alcohol—suggest the possibility of a cross-tolerance to alcohol and marihuana. None of the subjects used alcohol regularly, although they responded much the same as heavy drinkers might have. It is concluded that consideration of the dose, prior experience with drugs, setting, and possible cross-tolerance of alcohol and marihuana are important in evaluating the significance of the clinical effects.

638. Jordi, A.

VERGIFTUNGEN DURCH DEN KUNSTDÜNGER KALKSTICKSTOFF

(CALCIUMCYANAMID). [Poisoning caused by the artificial fertilizer, nitrolime (calcium cyanamide)].

Schweiz. Med. Wschr. (Basel), 188: 805-806 (6 ref.),

1947.

G – SEC – general – DC (sensit.) – humans – cardiovasc. – G.I. tract – nerv. syst. – metab. proc. – respir. – senses – skel., muscle, skin – unclass. ther. agents – *CAAAL-0 A-1290.

The author describes the mechanism and symptoms of calcium cyanamide poisoning. Calcium cyanamide is broken into calcium carbonate and cyanamide in the body. Cyanamide increases the toxicity of morphine, codeine, atropine, and strychnine, and also the diuretic action of caffeine and theobromine. It hinders the catalase system, hence slowing the synthesis of phenol to phenolglucuronic acid, and lowering the reducing power of tissue with respect to m-dinitrobenzol. Thus, cyanamide strongly influences the oxidation-reduction processes of the cell, presumably by affecting the sulfhydryl group of cysteine contained in glutathion. Calcium cyanamide alone is poisonous in quantity, but, with alcohol, the toxicity is increased by 30 times. Symptoms are: reddening of the upper body, headache or dizziness, deeper breathing, palpitation, and lower blood pressure. Calcium oxide, by forming calcium hydroxide, causes boils and skin irritation, and is often present in calcium cyanamide fertilizer. 3 cases of combined poisoning by alcohol and calcium cyanamide are described. Treatment is by injection of cysteine. Some prophylactic measures are described: mix fertilizer in something enclosed; do not spread against the wind; wear boots and tight-fitting clothes; cover unprotected skin with oil or vaseline; wash thoroughly after working; and take no alcohol before, during, or after work.

639. Jossierand, M.

THE ABILITY OF COPRINI TO SENSITIZE MAN TO ETHYL ALCOHOL.

Mycologia (New York), 44: 829-831 (3 ref.),

1952.

F – general – DC (sensit.) – humans – blood comp., sites, lymph – cardiovasc. – *CAAAL-0

A-1291.

In refutation of an article by George P. Child (Mycologia, 44: 200-202, 1952), which discusses the inability of *Coprinus atramentarius* to sensitize man to ethyl alcohol, stating that when *Coprinus atramentarius*, *C. comatus* and *C. micaeus* are consumed prior to ingestion of alcohol, there is no toxic effect (as is the case when *Panaeolus companulatus* is given), the author states that a toxic *Coprinus*-alcohol reaction is possible. *C. atramentarius*, at least in France, according to the author, frequently produces facial rubor and an acceleration of pulse rate, and the ingestion of alcohol reinforces the symptoms considerably. The identity of mushrooms in cases coming to the attention of the author were confirmed by a competent mycologist, and any possibility of accidental ingestion of *Panaeolus* was ruled out completely. Also, it was established that neither anaphylaxis, nor idiosyncrasy, nor a possible toxic mutation in *Coprinus* mycelia could explain the phenomenon. Repeated precipitation of symptoms following a *Coprinus* meal may be initiated by alcohol for up to 2 or 3 days. Not everyone is affected equally, since 1 person ate, with alcohol, the remains of a crop which had made 2 other persons ill, yet suffered no adverse effects. There is no satisfactory explanation to date for the reaction.

640. Jovanovic, U. J., Dürriegl, V., and Rogina, V.

EEG PATTERNS IN HEALTHY MEN AFTER EXPERIMENTAL APPLICATION OF PSYCHOTROPIC DRUGS AND ALCOHOL.

Electroenceph. Clin. Neurophysiol. (Amsterdam), 30: 269 (0 ref.), 1971.
 E – abst. – exp. cont. – exp. comp. – DC (unspec.) – humans – acute admin. – in vivo – blood lev.
 – psychol. perform. – CNS – senses – tranquilizers – *CAAAL-0 B-0941.

100 healthy, young, and middle-aged subjects of both sexes were examined electroencephalographically; additional acoustic and optic stimuli were applied, and the blood alcohol level (BAL) was checked. Following this, the subjects were divided into 2 large groups of 50 subjects, each group containing 3 subgroups. Subgroup 1a (10 subjects) received a placebo, subgroup 1b (20 subjects) 10 mg tacitin (a tranquilizer), and subgroup 1c (20 subjects) 20 mg tacitin. Subgroup 2a received a placebo plus alcohol (enough wine or spirits to achieve a BAL of 0.8-1.0%), and subgroup 2b 10 mg tacitin plus the same alcohol dose as 2a (subgroup 2c administration not mentioned). It was found that the basic EEG rhythm in subgroup 1a was hardly changed; in 1b and 1c, there was an increase in percentage of alpha waves. In 2a, 2b, and 2c, there was a stronger increase of the basic alpha rhythm, but also a decrease of wave frequency, and an increase of wave amplitude. Reaction times to simple acoustic and visual stimuli were unaffected in all subgroups. The psychiatric findings revealed variations dependent on individual compatibility to alcohol.

641. Joyce, C. R. B., Edgecombe, P. C. E., Kennard, D. A., Weatherall, M., and Woods, D. P.
POTENTIATION BY PHENOBARBITONE OF EFFECTS OF ETHYL ALCOHOL ON HUMAN BEHAVIOUR.
 Brit. J. Psychiat. (London), 105: 51-60 (15 ref.), 1959.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – mot. perform.
 – psychol. perform. – senses – barbiturates – *CAAAL-9415-J1 A-0819.

The complex reaction times of 8 healthy male subjects were observed before and at two intervals after ingesting the equivalent of 60 ml ethanol plus 130 mg phenobarbitone, the equivalent of 30 ml ethanol plus 65 mg phenobarbitone, or placebos. Mean reaction time decreased in all cases. 3 hr after treatment, the decrease after phenobarbitone was smaller than after placebo, but it was greater after alcohol or alcohol and phenobarbitone. "Although the effects of phenobarbitone were more striking than, and usually in the opposite direction from those of alcohol, the effect of consuming half doses of both drugs together was in the same direction as and larger than that of taking a whole dose of alcohol alone." The systematic trends observed were very unlikely to have occurred by chance (probability less than 0.01). The results support the belief that phenobarbitone potentiates some of the effects of ethanol.

642. Juul, P.
DIAZEPAMUM (VALIUM). II. DIAZEPAM SOM HYPNOTIKUM: 34 TILFAELDE AF FORGIFTNING. [Diazepam (valium). II. Diazepam as a hypnotic: 34 cases of poisoning].
 Ugeskr. Laeg. (Copenhagen), 128(4): 112-115 (0 ref.), 1966.
 Da – ES – SEC – stat. surv. – DC (add., infra-add., unspec. incr.) – humans – CNS – tranquilizers
 – *CAAAL-0 B-0330.

The use of diazepam as a hypnotic in 116 rheumatic patients is evaluated, and 34 cases of intoxication through the use of diazepam, alone or in combination with other substances, are described. 21 cases involved the combination of diazepam with other psychopharmacological drugs, sedatives, or alcohol, including 9 cases of coma. 3 cases of coma involved alcohol-diazepam combinations. It is concluded that diazepam taken alone is, by and large, atoxic, whereas the intensifying effects of combinations with other sedatives, hypnotics, and alcohol create extremely dangerous complications. Such possibilities must be borne in mind at the time of prescription.

643. Kahil, M. E., Cashaw, J., Simons, E. L., and Brown, H.
ALCOHOL AND THE TOLBUTAMIDE RESPONSE IN THE DOG.
 J. Lab. Clin. Med. (St. Louis), 64(5): 808-814 (27 ref.), 1964.

E – exp. cont. – DC (sensit.) – mammals – acute admin. – chronic admin. – in vivo – blood lev. – absorp., distrib., stor. – liver, kidney – metab. proc. – diagnost. agents – enzymes – hormones, hormone antag. – unclass. ther. agents – *CAAAL-0 A-1406.

Starvation for 7 days, followed by 3 weeks of undernutrition, did not affect a consistent blood glucose fall after 10 mg/kg tolbutamide was given iv to dogs. When alcohol was given daily (3 g/kg, 20% sol by stomach tube) for 28 days, the fall in blood sugar at 1 and 2 hr after tolbutamide was not affected; but, at 6 and 9 hr, the blood glucose levels were significantly lower. Counter-regulatory mechanisms to hypoglycemia were evaluated on the basis of urinary indicators of adrenal cortical response. After a period of alcohol ingestion, there was an increased excretion of 17-ketogenic steroids on the day of sustained hypoglycemia following tolbutamide challenge. A moderate decrease in catecholamine hypoglycemia occurred after 3 weeks of alcohol administration; however, following tolbutamide-induced hypoglycemia, there was a further and unexpected fall in excretion of 3-methoxy-4-hydroxymandelic acid. This apparent failure of counter-regulatory mechanisms may involve renal blood flow. Liver biopsies and the bromsulphalein test indicated no change in liver structure or function. The glucagon test showed no effect of alcohol on glycogen stores. It is suggested that prolonged alcohol ingestion is accompanied by a decreased catecholamine excretion, and that the sustained tolbutamide-induced hypoglycemia in dogs subjected to undernutrition and alcohol intake occurs concomitantly with a decrease in catecholamine output.

644. Kalant, H., Hawkins, R. D., and Watkin, G. S.

THE EFFECT OF ETHANOL ON THE METABOLIC RATE OF RATS.

Canadian Journal of Biochemistry and Physiology (Ottawa), 41: 2197-2203 (14 ref.), 1963.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – CNS – metab. proc. – autonomic agents – enzymes – *CAAAL-10909-B2 A-0820.

Ethanol was administered ip to 8 male rats in doses of 2 and 4 g/kg in a 20% saline sol. Glucose and saline controls were used. Control readings of the oxygen consumption started 30 min before immersion in a constant temperature bath of 27°C, and were taken for 30 min before injection and for 90 min thereafter. It was found that the 0.4 g/kg ethanol dose caused a small but significant elevation in the metabolic rate of anaesthetized rats; the effect was masked in unanaesthetized rats. Pretreatment with iproniazid (100 mg/kg sc) or phenoxybenzamine (10 mg/kg ip) failed to enhance or block the initial elevation of oxygen consumption induced by ethanol. It is suggested that the initial increase in oxygen consumption is caused by some other mechanism than the release of adrenaline or noradrenaline.

645. Kalant, H., and Grose, W.

EFFECTS OF ETHANOL AND PENTOBARBITAL ON RELEASE OF ACETYLCHOLINE FROM CEREBRAL CORTEX SLICES.

J. Pharmacol. Exp. Ther. (Baltimore), 158(3): 386-393 (33 ref.), 1967.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vitro – dose resp. – CNS – barbiturates – *CAAAL-0 B-0331.

Ethanol reduced the amount of acetylcholine released by slices of rat or guinea pig cerebral cortex. Low concentrations of pentobarbital stimulated, and high concentrations inhibited, acetylcholine release. Both stimulation and inhibition were increased by raising the potassium ion (K^+) concentration of the medium. Brain slices from animals made tolerant to ethanol were refractory to the in vitro effect of ethanol on acetylcholine release. The interaction of ethanol and pentobarbital in combination did not appear to be simply additive, since 0.11 M ethanol decreased acetylcholine release in the presence of 5 mM K^+ , and had no effect in 15 mM K^+ , yet significantly increased the stimulatory effect of 0.01 mM pentobarbital in 15 mM K^+ .

646. Kalant, H., Khanna, J. M., and Marshman, J.
 EFFECT OF CHRONIC INTAKE OF ETHANOL ON PENTOBARBITAL METABOLISM.
 J. Pharmacol. Exp. Ther. (Baltimore), 175(2): 318-324 (31 ref.), 1970.
 E – exp. cont. – cross-tol. – mammals – acute admin. – chronic admin. – in vivo – in vitro – blood
 lev. – mot. perform. – absorp., distrib., stor. – CNS – metab. proc. – barbiturates – *CAAAL-0
 B-0942.

To investigate the effects of chronic ethanol intake on the distribution and central effects of pentobarbital, as well as on its metabolism under conditions approximating those in vivo, ethanol was fed to rats for 2 weeks at 5.1% w/v of a liquid diet (10-12 g ethanol/kg/day) up to 24 hr before an ip pentobarbital sodium injection (30 mg/kg). Induction and sleeping times were based on righting reflex. Ethanol reduced the times of onset and duration, and sleeping time after pentobarbital; at 15 min after injection, the pentobarbital concentrations in plasma and brain were lower in the ethanol group (although the distribution between blood and brain was almost the same in the group given to ethanol). At 30 and 60 min, the difference between the 2 groups disappeared. The lack of metabolic effect was confirmed by in vitro tests on liver slices. Rats were given phenobarbital sodium 100 mg/kg/day by intubation for 1 week. This pretreatment significantly increased the pentobarbital uptake of liver slices. In contrast, ethanol pretreatment caused a net uptake of 33% less than controls. Results are consistent with reports on cross-tolerance cited. Alcohol appears to achieve its effect more by reducing sensitivity of the CNS, than by affecting the metabolism of pentobarbital.

647. Káldor, A., and Pogátsa, G.
 ZUR WIRKUNG DES CARBUTAMIDS AUF DAS ZENTRALNERVENSYSTEM. [The
 effect of carbutamide on the central nervous system].
 Klin. Wschr. (Berlin), 38: 1114-1116 (11 ref.), 1960.
 G – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin.
 – in vivo – blood lev. – CNS – respir. – hormones, hormone antag. – *CAAAL-0 A-1292.

A series of experiments were performed to determine the influence of carbutamide on the toxicity of alcohol. The toxicity of 20% alcohol, alone and in combination with carbutamide, was determined. Carbutamide (500 mg/kg) was given to mice 2 hr before the administration of 4.0 g/kg alcohol ip, and the results were statistically evaluated. Alcohol-carbutamide was found to be 1.43 times more toxic than alcohol alone. Experiments were also conducted to determine the influence of alcohol and carbutamide on the blood sugar level. 0.5 g of carbutamide and 4.09 g/kg of alcohol were administered, alone and together, to rats. Alcohol influenced neither the blood sugar concentration nor the effect of carbutamide. In other experiments on the combined effect of alcohol and carbutamide on respiration, body temperature, and narcosis (performed on rabbits and mice), it was found that respiration was significantly decreased. The narcotic effect of ethanol was significantly enhanced by carbutamide. It is concluded that alcohol increases the toxic effect of carbutamide, and this can be partially explained by the combined effect on the CNS.

648. Kalsner, S.
 THE POTENTIATING EFFECTS OF ETHANOL ON RESPONSES OF AORTIC STRIPS
 TO STIMULANT DRUGS.
 J. Pharm. Pharmacol. (London), 22(11): 877-879 (8 ref.), 1970.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vitro – mot.
 perform. – skel., muscle, skin – diagnost. agents – tranquilizers – *CAAAL-0 B-0943.

Strips of rabbit aorta were contracted, on the sharply-rising portions of the drug dose response curves, by noradrenaline, methoxamine, histamine, and potassium. With strips exposed to 174 mM ethanol, at the stable plateau response values of the drugs, the augmentation response to 3×10^{-9} g/ml noradrenaline, 5×10^{-8} g/ml methoxamine, and 5×10^{-8} g/ml histamine was equivalent to doubling the agonists in the muscle chambers, but contractions induced by 20mM potassium were increased

less by ethanol than by an equivalent concentration of potassium alone. In vivo pretreatment with 1 mg/kg reserpine (in 10% ascorbic acid, 18-24 before death) failed to modify the response to ethanol, as did pretreatment of aortic strips with reserpine and phenoxybenzamine (1×10^{-6} g/ml for 10 min). Ethanol at 348 mM exerted direct contractile effects which at 174 mM were barely seen. Soaking strips in disodium EDTA did not affect the potentiating effect of ethanol on the response to noradrenaline. However, the direct contractile effect of ethanol in the calcium-free sol was decreased to about 12%, as was the response to noradrenaline. Mechanisms are discussed.

649. Kane, R. L., Talbert, W., Harlan, J., Sizemore, G., and Cataland, S.
A METHANOL POISONING OUTBREAK IN KENTUCKY.
 Arch. Environ. Health (Chicago), 17: 119-129 (55 ref.), 1968.
 E – SEC – case hist. – DC (antidotal) – post-mort. – humans – blood lev. – other drug lev. – metab. proc. – alcohols – *CAAAL-13465 B-0528.

The use of ethanol in the treatment of acute methanol poisoning is evaluated in this clinical epidemiological study. 18 known cases of poisoning were attributed to the consumption of a shellac solvent diluted to about 37 vol % methanol. There were 8 deaths, including 1 of the 11 victims who were treated in hospital. Because of the experimental and clinical efficacy of ethanol in delaying methanol metabolism, together with its tendency to promote water diuresis even in the face of dehydration, ethyl alcohol therapy was attempted. However, adequate blood levels of ethanol (100 mg/100 cc) were obtained in only 1 patient after admission, due to inadequate loading doses. It was observed that the 5 patients with blood ethanol levels (118-346 mg/100 cc) on admission had significantly less acidosis (pH 7.30-7.58) than the 6 patients who had no ethanol in their blood (pH 7.04-7.55). In support of the protective effect of ethanol, all blood determinations in fatal cases showed no ethanol.

650. Kaplan, H. L., Forney, R. B., Richards, A. B., and Hughes, F. W.
DEXTRO-AMPHETAMINE, ALCOHOL, AND DEXTRO-AMPHETAMINE-ALCOHOL COMBINATION AND MENTAL PERFORMANCE.
 In: Harger, Rolla N., ed. *Alcohol and Traffic Safety*. Proceedings of the Fourth International Conference on Alcohol and Traffic Safety at Indiana University, December 6-10, 1965. Bloomington, Indiana: Indiana University Press, pp. 211-214 (0 ref.), 1966.
 E – exp. cont. – presentation – DC (decrease) – humans – acute admin. – in vivo – blood lev. – other drug lev. – psychol. perform. – amphetamines – *CAAAL-0 B-0332.

In a double-blind study, four groups, each containing 10 human subjects, were treated with one of the following: 1) placebo drug, 2) d-amphetamine (5 mg) 3) placebo drug plus alcohol, and 4) d-amphetamine (5 mg) plus alcohol. D-amphetamine improved the mental performance over placebo throughout the four-hour experiment. The effect of the d-amphetamine-alcohol combination did not differ much from that of placebo plus alcohol. In initial tests, performance with alcohol-d-amphetamine was significantly better than with alcohol-placebo, but was significantly impaired when compared with d-amphetamine alone. Alcohol, therefore, suppressed the performance-enhancing action of the drug.

651. Kaplan, H. L., Forney, R. B., Hughes, F. W., Richards, A. B., and Jain, N. C.
ETHANOL EFFECTS ON CHLORAL HYDRATE METABOLISM IN MICE.
 Toxicol. Appl. Pharmacol. (New York), 10(2): 387 (0 ref.), 1967.
 E – abst. – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – other drug lev. – CNS – metab. proc. – sed., hypnot. – *CAAAL-0 B-0333.

Male mice received ethanol (4 g/kg, intragastric), chloral hydrate (0.5 g/kg, intragastric), or both in combination. At various times after administration, the mice were sacrificed and chloral hydrate, trichloroethanol, and ethanol content determined by gas chromatography. Comparison of drugs and

metabolites showed that a slower rate of degradation of ethanol and trichloroethanol occurred after ethanol plus chloral hydrate. The synergism appeared to be one of mutual interaction—not only was ethanol metabolism inhibited by chloral hydrate, but the trichloroethanol levels were prolonged.

652. Kaplan, H. L., Forney, R. B., Hughes, F. W., and Jain, N. C.
CHLORAL HYDRATE AND ALCOHOL METABOLISM IN HUMAN SUBJECTS.
J. Forensic Sci. (Mundelein), 12(3): 295-304 (5 ref.), 1967.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev.
 – cardiovasc. – CNS – metab. proc. – sed., hypnot. – *CAAAL-0 B-0334.

5 male students received, on different occasions, 0.88 g/kg ethanol, 1.0 g chloral hydrate, or both in combination. It was found that ethanol metabolism was not appreciably altered by chloral hydrate administered simultaneously; however, chloral hydrate metabolism was markedly altered—blood trichloroethanol concentrations reached earlier and higher peak levels, and remained elevated during the remainder of the 6 hr period. The number and severity of symptoms were greater after the combination than after either compound alone, but no “knock-out” effect was observed.

653. Kaplan, H. L., Jain, N. C., Forney, R. B., and Richards, A. B.
CHLORAL HYDRATE-ETHANOL INTERACTIONS IN THE MOUSE AND DOG.
Toxic. Appl. Pharmacol. (New York), 14: 127-137 (8 ref.), 1969.
 E – exp. cont. – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
 – in vivo – dose resp. – other drug lev. – absorp., distrib., stor. – blood comp., sites, lymph – CNS
 – metab. proc. – anesthetics – *CAAAL-0 B-0944.

Male Swiss albino mice were given 0.5 g/kg chloral hydrate intragastrically, 4 g/kg ethanol intragastrically or ip, or both drugs in combination. Sleeping time was the index for depressant action. As well, whole mice were homogenized for analysis of metabolites. The mean chloral hydrate-induced sleeping time was increased more than 5-fold by concurrent administration of ethanol. Chloral hydrate metabolism was not significantly altered by ethanol, but trichloroethanol concentrations were significantly greater after combined administration. A marked inhibition of ethanol metabolism by chloral hydrate was also evident. The substantial enhancement of depressant effect was not found in 4 dogs given 0.5 g/kg chloral hydrate plus 1.5 g/kg ethanol, nor was chloral hydrate metabolism altered. However, blood trichloroethanol concentrations rose more rapidly and obtained a greater peak with concurrent doses. The interaction between chloral hydrate and ethanol in dogs may involve an inhibition of the oxidation of chloral hydrate to trichloroacetic acid, resulting in its greater availability for reduction to trichloroethanol.

654. Kastor, O., and Horáček, J.
EIN AKUT TÖDLICH VERLAUFENDES MUKO-KUTANEO-OKULARES SYNDROM BEI EINEM 35 JÄHRIGEN MANN NACH BEHANDLUNG MIT 2-SULFANILAMIDO-6-METHOXYPYRIMIDIN (SULFAMETHOXYDIN). [An acute muco-cutaneo-ocular syndrome with lethal result in a 35 year-old man after treatment with 2-sulfanilamido-6-methoxypyrimidine (sulfamethoxydin)].
Derm. Wschr. (Leipzig), 153(48): 1318-1323 (8 ref.), 1967.
 G – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – respir. – senses – skel., muscle, skin – analg., antipyret. – anti-infectants – *CAAAL-0 B-0335.

Case material is given concerning a 35 yr-old man taken ill with angina, and treated with aureomycin and sulfamethoxydin (total dose 5 g). Following this treatment, the patient consumed about 250 ml alcohol at a party, and later took an amidopyrine analgesic for ensuing headache. A muco-cutaneo-ocular syndrome developed the next day, terminating in death 48 hr later, in spite of corticoid and antibiotic therapy. The authors conjecture that the lethal effect was due to the cumulative incompatibility of amidopyrine and alcohol.

655. Kater, R. M. H., Zeive, P., Tobon, F., Roggin, G., and Iber, F. L.
ACCELERATED METABOLISM OF DRUGS IN ALCOHOLICS.
 Gastroenterology (Baltimore), 56(2): 412 (0 ref.), 1969.
 E – abst. – exp. cont. – cross-tol. – drug-dep. humans – blood lev. – metab. proc. – hormones, hormone
 antag. – anticonvulsants – coagulants – *CAAAL-0 B-0336.

Heavy-drinking alcoholics (200 g or more/day) were compared with non-drinking control subjects as to the rate of removal of three microsomally metabolized drugs from the body: tolbutamide, warfarin, and dilantin. 1 g tolbutamide was administered iv and blood samples collected. In control subjects, the mean half-life was 350.6 min, and in alcoholics the mean half-life was 165.4 min. Warfarin, given in a single dose of 40 mg po, had a mean half-life of 26.5 hr in alcoholics, compared to 41.0 hr for controls. Dilantin was administered 3 times daily, 100 mg for 3 days, and had a mean half-life of 16.3 hr in alcoholics, compared to 23.5 hr in controls. It is concluded that the heavy use of alcohol stimulates the metabolism of a variety of microsomally-metabolized drugs to a clinically important degree.

656. Kater, R. M. H., Roggin, G., Tobon, F., Zeive, P., and Iber, F. L.
INCREASED RATE OF CLEARANCE OF DRUGS FROM THE CIRCULATION OF ALCOHOLICS.
 Amer. J. Med. Sci. (Philadelphia), 258: 35-39 (13 ref.), 1969.
 E – exp. cont. – exp. comp. – cross-tol. – drug-dep. humans – acute admin. – chronic admin. – in
 vivo – blood lev. – liver, kidney – metab. proc. – anticonvulsants – coagulants – hormones, hormone
 antag. – *CAAAL-14498 B-0572.

The increased rates of clearance from the circulation of tolbutamide, warfarin, and diphenylhydantoin were studied in 61 alcoholics. All alcoholic subjects had been drinking heavily (250 g or more of alcohol/day) for at least 3 months, but drinking was stopped and alcohol had disappeared from the blood prior to drug administration. The mean plasma half-life of 1 g of tolbutamide sodium, administered iv, was found to be 165.4 min in 31 alcoholics, and 350.6 min in 13 abstinent control subjects. The half-life of 40 mg of warfarin sodium, administered po, was 26.5 hr in 15 alcoholics, and 41.1 hr in 11 controls. After administration of 100 mg diphenylhydantoin 3 times/day for 3 days, the half-life was 16.3 hr in 15 alcoholics, and 23.5 hr in 76 controls. It is concluded that the metabolism of tolbutamide, warfarin, and diphenylhydantoin is increased in humans after chronic alcohol ingestion by a nonspecific induction of hepatic microsomal enzymes similar to that known to occur in rats.

657. Kater, R. M. H., Tobon, F., and Iber, F. L.
INCREASED RATE OF TOLBUTAMIDE METABOLISM IN ALCOHOLIC PATIENTS.
 J.A.M.A. (Chicago), 207(2): 363-365 (8 ref.), 1969.
 E – exp. cont. – cross-tol. – drug-dep. humans – acute admin. – in vivo – blood lev. – liver, kidney
 – metab. proc. – hormones, hormone antag. – *CAAAL-13361-C10 B-0529.

An increased rate of tolbutamide metabolism was studied in 20 alcoholic patients who had each consumed at least 250 g of alcohol/day for 2 years or more. After sufficient time was allowed for alcohol to be cleared from the body, 1 g of tolbutamide was injected iv. Venous samples were collected, and serum tolbutamide levels determined at 30 and 60 min and then hourly for 6 hr. The mean biological half-life for tolbutamide was found to be 165.4 min (standard deviation (SD) ± 33.5) in the alcoholics, as compared to 350.6 min (SD ± 130.6) in 10 non-alcoholic controls. A highly significant increase (probability less than 0.005) in the disappearance rate of tolbutamide was observed in the serum of the alcoholics. The authors conclude that it is reasonable to speculate that the prolonged heavy intake of alcohol increases the rate of tolbutamide metabolism by an induction of hepatic microsomal drug-metabolizing enzymes similar to that described in experimental animals.

658. Kater, R. M. H., Zeive, P., Tobon, F., Roggin, G., and Iber, F. L.
ACCELERATED METABOLISM OF DRUGS IN ALCOHOLICS.
 Gastroenterology (Baltimore), 56: 412 (0 ref.), 1969.
 E – abst. – exp. cont. – cross-tol. – humans – drug-dep. humans – acute admin. – chronic admin.
 – in vivo – blood lev. – liver, kidney – metab. proc. – *CAAAL-0 B-0945.

The effect of chronic alcohol use on the metabolism of 2 microsomally-metabolized drugs was studied in alcoholic subjects who had consumed a min of 200 g ethanol/day for at least 3 weeks prior to admission, and was compared to drug metabolism in control subjects who were abstinent (60%), or who consumed less than 10 g ethanol/day (40%). Alcoholics were studied within 8 days of cessation of drinking, and usually within 3 days. After administration of 1 g tolbutamide iv, and collection of blood samples hourly for 6 hr, it was found that the mean half-life was 350.6 min in 10 control subjects, and 165.4 min in 20 alcoholics. After 40 mg warfarin po, and collection of blood samples 12, 24, 48, and 96 hr after administration, the mean half-life was 41.0 hr in 10 control subjects, and 26.5 hr in 15 alcoholics. After 3 x 100 mg dilantin po/day for 3 days, the collection of samples at 12, 24, 48, 96, and 120 hr showed a mean half-life of 23.5 hr in 76 control subjects, and of 16.3 hr in 15 alcoholics. All differences were significant. It is concluded that heavy alcohol consumption stimulates the metabolism of a variety of microsomally-metabolized drugs to a clinically important degree.

659. Katkin, E. S., and Hayes, W. N.
DIFFERENTIAL EFFECTS UPON REACTION TIME AND PERCEPTUAL MOTOR PERFORMANCE OF ALCOHOLIC BEVERAGES DIFFERING IN CONGENER CONTENT.
 In: Garvey, W. D., et al., eds. *Proceedings of the 75th Annual Convention of the American Psychological Association, 1967.* Washington: American Psychological Association, pp. 9-10 (2 ref.), 1967.
 E – exp. cont. – exp. comp. – presentation – congen. stud. – humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – *CAAAL-0 B-0337.

Simple and complex reaction time tests were performed by 18 fasting students, before and after drinking 4 doses of 0.4 ml alcohol (as bourbon or vodka containing 43% alcohol)/kg or water, at 1 hr intervals. At blood alcohol concentrations of 0.07%, performance was significantly lower after either beverage than in controls. Mirror drawing scores were significantly lower after bourbon than after water, but scores after vodka were not significantly different from those after water. The results suggest that alcohol determines the immediate effects on reaction time; the congeners, found in bourbon but not in vodka, determine the long-term reaction time effects.

660. Katkin, E. S., Hayes, W. N., Teger, A. I., and Pruitt, D. G.
EFFECTS OF ALCOHOLIC BEVERAGES DIFFERING IN CONGENER CONTENT ON PSYCHOMOTOR TASKS AND RISK TAKING.
 Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 101-114 (11 ref.), 1970.
 E – exp. – congen. stud. – humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – CNS – *CAAAL-12892 B-0530.

The effects of bourbon whiskey (high congener content) and vodka (low congener content) on psychomotor performance and on risk-taking were studied in human subjects. In a double blind procedure, 18 subjects were administered, over a 4 hr period, 1.6 ml/kg water, or ethanol in the form of either 86 proof bourbon or vodka. Psychomotor tasks and breath analysis for blood alcohol levels were performed before, and at 1 and 5 hr after, drinking. The results indicate that the immediate effects of alcoholic beverages (at 1 hr) on reaction time were determined by the ethanol, and that the congener effects were minimal. After the ethanol was metabolized (at 5 hr), the bourbon, i.e., its congeners, showed a greater deleterious effect, especially in decision-making. In a second experiment, 36 men completed a 12-item choice-dilemma questionnaire, before and 1/2 hr after drinking 0.8 ml/kg

ethanol as either bourbon, vodka, or ethanol. The results indicated a greater tendency to risk-taking after bourbon. It is concluded that congeners impair psychomotor performance, and increase risk-taking behavior.

661. Kato, R.

UN PRETRATTAMENTO, ESEGUITO 48 ORE PRIMA, CON SVARIATE SOSTANZE PUÒ DIMINUIRE GLI EFFETTI FARMACOLOGICI DEL NEMBUTAL. [A pretreatment performed 48 hours previously with different substances which diminish the pharmacologic effects of nembutal].

Società Lombarda di Scienze Mediche e Biologiche, Atti (Milan), 14: 777-780 (7 ref.), 1959.
I – ES – SEC – exp. cont. – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – CNS – barbiturates – *CAAAL-0 A-0821.

A series of drugs were tested in rats to find antagonists to pentobarbital intoxication. The following substances, given 48 hr prior to pentobarbital (nembutal) administration, proved to be effective to some degree in reducing the toxicity: phenoglycodol, pentothal, luminal, doriden, nembutal, evipan, meprobamate, chlorpromazine, chloreton, urethane, phenylbutazone, diphenylhydantoin, aminopyrine. The following substances, on the other hand, had little effect: promazine, tofranil, atarax, chlorcyclizine, mysoline, viadril, chloral hydrate, alcohol, 3,4-benzpyrine, and 3-methylcholantrene. 12 cc/kg 40% alcohol ip was administered to 7 control rats and to 6 rats pretreated with 25 mg/kg nembutal ip. Sleeping time for the controls was 88 ± 8.7 min, and for the nembutal-treated rats was 87 ± 6.0 min; thus, the effect of nembutal pretreatment was insignificant.

662. Kato, R., and Chiesara, E.

INCREASE OF PENTOBARBITONE METABOLISM INDUCED IN RATS PRETREATED WITH SOME CENTRALLY ACTING COMPOUNDS.

Brit. J. Pharmacol. (London), 18: 29-38 (15 ref.), 1962.
E – SEC – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – in vitro – blood lev. – other drug lev. – CNS – liver, kidney – metab. proc. – barbiturates – *CAAAL-0 A-1407.

The effects of pretreatment with 31 centrally-acting drugs on the metabolism of pentobarbitone were determined in rats. Tests were made on 160-180 g female rats, although, in some vitro tests, 60 g male rats were used. 48 hr after ip administration of 1 of the drugs, various doses of pentobarbitone were injected ip. Sleeping times were measured, and, 1 hr later, the animals were killed and the pentobarbitone concentration determined in serum and brain. Liver enzyme activity was also determined in liver slices. It was found that pretreatment with ethyl alcohol (dosage unstated) failed to influence sleeping time after 25 mg/kg pentobarbitone or pentobarbitone concentrations, nor did it affect the in vitro enzymatic activity of liver slices. The general results are in accord with the view that the capacity of compounds to increase pentobarbitone metabolism may be related to their ability to act directly on microsomal enzyme systems.

663. Kato, R., and Vassanelli, P.

INDUCTION OF INCREASED MEPROBAMATE METABOLISM IN RATS PRETREATED WITH SOME NEUROTROPIC DRUGS.

Biochem. Pharmacol. (New York), 11: 779-794 (19 ref.), 1962.
E – SEC – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – in vitro – blood lev. – other drug lev. – absorp., distrib., stor. – liver, kidney – metab. proc. – nerv. syst. – tranquilizers – *CAAAL-0 A-1408.

The effects of ip administration of more than 35 drugs—hypnotics, anticonvulsants, tranquilizers, central myorelaxants, and central stimulants—on the metabolism of meprobamate were determined in rats. Tests were made on 160-180 g female rats, although, in some in vitro tests, 60 g male rats

were used. 48 hr after pretreatment with 1 of the drugs, rats were injected with meprobamate ip. 3 hr after the meprobamate, the animals were sacrificed and meprobamate measured in serum and brain. It was found that pretreatment with 10 ml/kg 40% ethanol sol failed to accelerate the metabolism of 150 mg/kg meprobamate, nor did ethanol stimulate activity of meprobamate-metabolizing liver enzymes. The general results indicate a possible role of the metabolic factor in the development of tolerance and cross-tolerance to meprobamate in animals and in clinical experiments.

664. Kawahara, M.

IWAYURU KÔSEISHIN SHINKEIZAI NO KETCHÛ ARUKÔRU-CHI NI OYOBOSU

EIKYÔ NI TSUITE. [The influence of so-called psycho- and neurotropic drugs upon the alcohol metabolism].

Acta Med. (Igaku Kenkyu) (Fukuoka), 31: 957-978 (50 ref.),

1961.

J – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – analg., antipyret. – anticonvulsants – autotoxins – autonomic agents – stimulants – tranquilizers – *CAAAL-9911-A2 A-0822.

Groups of 5 to 8 rabbits were given im injections of the following: epinephrine, 1 mg; pilocarpine, 10 mg; sodium phenylbutazone, 300 mg; diphenhydramine, 30 mg; reserpine, 1 mg; tetraethylammonium bromide, 100 mg; 2-benzyl-imidazoline hydrochloride, 40 mg; finalin, 20 mg; or chlorpromazine, 12.5 mg; followed in 30 min by 10 ml 25% ethanol/kg. Also, phenacetin (1.25 to 1.45 g), phenytoin (100 mg), meprobamate (2,500 to 3,300 mg), and pipradrol (240 to 320 mg), were given po 60 min before ethanol. Blood alcohol measurements taken 30 to 300 min after ethanol administration showed increased blood alcohol levels with all drugs except epinephrine and reserpine. The rates of decrease of blood alcohol in the treated animals were either parallel to or less than those of controls.

665. Kaye, S., and Haag, H. B.

STUDY OF DEATH DUE TO COMBINED ACTION OF ALCOHOL AND PARALDEHYDE IN MAN.

Toxic. Appl. Pharmacol. (New York), 6: 316-320 (13 ref.),

1964.

E – exp. – general – DC (add., infra-add., unspec. incr.) – drug-dep. humans – mammals – acute admin. – in vivo – blood lev. – CNS – sed., hypnot. – *CAAAL-0 A-0823.

Reported are 9 cases in which the patient, otherwise in good health except for alcoholism symptoms, died suddenly and unexpectedly following paraldehyde therapy for acute alcoholism. Death occurred from 1/2 hr to 4 hr after administration of paraldehyde (30-60 ml), and autopsies revealed no morphological cause of death. It appears that paraldehyde alone may produce death when the dose exceeds 120 ml, but the developing depression (coma) usually precedes death by at least 12 hr. The possible synergism which occurred in these cases appears to be confirmed by experiments on male albino mice which were fasted and then given 3/4 LD₅₀ alcohol po (LD₅₀ for mice—11.1 ml 95% alcohol/kg), followed 0, 2, 3, 4, 5, or 6 hr later by 1.79 ml/kg (LD₅₀) paraldehyde. The results indicated that there may be an additive synergism which becomes more pronounced when the interval between drugs is increased to several hr; however, synergism was only demonstrated at 5 hr (probability = 0.05).

666. Kaymakçalan, S., and Tuğrul, S.

TOLBUTAMIDE'İN ALKOL VE BARBITÜRALARIN TAVŞANDAKİ HIPNOTİK TESİRİ ÜZERİNE ETKİSİ. [The potentiation of the hypnotic effects of alcohol and barbiturates in rabbits by tolbutamide].

Türk İjiyen ve Tecrübî Biyoloji Dergisi (Istanbul), 24: 63-70 (14 ref.),

1964.

T – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – mot. perform. – CNS – liver, kidney – hormones, hormone antag. – *CAAAL-0

A-1335.

Experiments were conducted to determine the effect of tolbutamide on the hypnotic effect of alcohol, pentobarbital, and thiopental. Rabbits received 200 mg/kg tolbutamide by stomach tube for 15 days, followed by a single dose of 5 mg 70% alcohol/kg, and the sleeping times were determined. For tolbutamide-pretreated rabbits, the sleeping time was found to be 134.2 ± 11.0 min, as compared to 81.6 ± 2.4 min in a control group which had received no tolbutamide. The increase in sleeping time in the former group was 51.7%, a statistically significant rise at the less than 0.001 level of probability. Tests with barbiturates had similar results. To explain this increase, the authors hypothesize an inhibitory action of tolbutamide on liver enzymes, or a blockade of microsomal enzymes responsible for alcohol metabolism. It is concluded that conjunctive use of alcohol and tolbutamide by patients is hazardous, and that the possibility of a combined effect of alcohol and antidiabetic agents is of forensic importance.

667. Keeser, E.

IST ETWAS ÜBER KUMULATIVE WIRKUNG BEI GLEICHZEITIGEM GENUSS VON ALKOHOL UND TABAK BEKANNT? [Is something known about the cumulative effects of simultaneous use of alcohol and tobacco?].

Med. Welt (Stuttgart), 14: 611 (0 ref.),

1940.

G – general – DC (add., infra-add., unspec. incr.) – humans – cardiovasc. – CNS – G.I. tract – liver, kidney – *CAAAL-0 A-0824.

In reply to a question, it is stated that alcohol and nicotine affect, at least partially, the same organs and systems. Nicotine can decrease coronary blood flow, and cause myocardial damage, gastritis, neuritis, vasomotor disturbances, and ataxia. Alcohol can cause gastritis, heart damage, neuritis, ataxia, etc. It is therefore to be expected that simultaneous use of alcohol and tobacco can increase the effects. The author cannot say whether the 2 agents have a potentiative or an additive effect.

668. Kellogg, J. H.

THE RELATION OF TOBACCO USING AND OTHER DRUG HABITS TO ALCOHOLIC INTEMPERANCE.

Modern Medicine (Battle Creek), 8(6): 137-139 (0 ref.),

1899.

E – general – conj. addict. – drug-dep. humans – cardiovasc. – analg., antipyret. – *CAAAL-0

A-0825.

The author (a temperance advocate) states that the use of tobacco begins first, and then creates a demand for the use of alcohol. He considers it necessary to stop all other drug habits if a person is to have a chance to stop the alcohol habit, for these drugs are frequently used interchangeably. The author has encountered a number of cases in which alcohol and morphine, or morphine and cocaine were so used; in the case of 1 woman, large quantities of strong mocha coffee were a convenient substitute for alcohol. In many cases, the tobacco habit is clearly the fundamental vice, and the alcohol habit an addition to accentuate the pleasure from tobacco use, or to provide a temporary antidote for its toxic effects.

669. Kellogg, J. H.

THE RELATION OF TOBACCO USING AND OTHER DRUG HABITS TO ALCOHOLIC INTEMPERANCE.

Dietetic and Hygienic Gazette (New York), 15: 522-524 (0 ref.),

1899.

E – general – conj. addict. – drug-dep. humans – cardiovasc. – analg., antipyret. – *CAAAL-0

A-0826.

The author (a temperance advocate) states that the use of tobacco begins first, and then creates a demand for the use of alcohol. He considers it necessary to stop all other drug habits if a person is to have a chance to stop the alcohol habit, for these drugs are frequently used interchangeably. The

author has encountered a number of cases in which alcohol and morphine, or morphine and cocaine were so used; in the case of 1 woman, large quantities of strong mocha coffee were a convenient substitute for alcohol. In many cases, the tobacco habit is clearly the fundamental vice, and the alcohol habit an addition to accentuate the pleasure from tobacco use, or to provide a temporary antidote for its toxic effects.

670. Kelly, J. A.
TREATMENT OF CARBOLIC-ACID POISONING BY ALCOHOL, WITH REPORT OF A CASE.
Merck's Archives (New York), 1: 441 (1 ref.), 1899.
E – general – case hist. – DC (antidotal) – humans – skel., muscle, skin – anesthetics – gastrointest. agents – *CAAAL-0 A-0827.

A child (21 months old) swallowed 1 oz of carbolic acid, and spilled another oz over its face and chest. The treatment was as follows: 6 to 8 fluid drops of pure alcohol, followed by apomorphine (1/30 grain) 6 min later, then 1 fluid drop of undiluted whiskey every ten min for 8 doses. By the next day, the child was normal. It is concluded that alcohol is an absolute antidote for carbolic acid, and lavage of the stomach with alcohol should be resorted to in preference to any other method. Where lavage is not practicable, alcohol or whiskey, preferably the former, should be given, followed by apomorphine as an emetic.

671. Kendal, L. P., and Ramanathan, A. N.
LIVER ALCOHOL DEHYDROGENASE AND ESTER FORMATION.
Biochem. J. (London), 52: 430-438 (14 ref.), 1952.
E – exp. cont. – DC (decrease) – mammals – in vitro – liver, kidney – metab. proc. – alcohols – *CAAAL-0 A-0828.

Questioned was the opinion that the toxic effects of methanol in man are minimized by the simultaneous ingestion of ethanol. In vitro tests provided evidence of competitive inhibition by ethanol of the oxidation of methanol to formaldehyde by liver alcohol dehydrogenase. The behaviour of formaldehyde, alone and in the presence of methanol and ethanol, was examined in mutase-containing alcohol dehydrogenase preparations. When methanol was present in the system, the disappearance of formaldehyde was accelerated, and the appearance of formate was increased correspondingly. In the presence of ethanol, total formate never accounted for more than half of the formaldehyde which disappeared. A significant fraction of the oxidation of coenzyme I H_2 by formaldehyde to give methanol was balanced by a reduction of coenzyme I by ethanol to give coenzyme I H_2 and acetyldehyde.

672. Kendal, L. P., and Ramanathan, A. N.
EXCRETION OF FORMATE AFTER METHANOL INGESTION IN MAN.
Biochem. J. (London), 54: 424-426 (12 ref.), 1953.
E – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – other drug lev. – metab. proc. – alcohols – *CAAAL-0 A-0829.

The effects of ethanol on the excretion of formate from the body after methanol ingestion were investigated. 2 adult males were given 0.05 g/kg methanol po, diluted in 100 ml water, immediately after the bladders were emptied. In 1 experiment, the subjects were given no ethanol; several weeks later, another experiment was conducted on the same subjects, who were then given 15 ml ethanol simultaneously with the methanol, followed by 10 ml ethanol every hr. Urine was collected over a period of 12 hr, and the methanol and formate concentrations determined. It was found that the appearance of formate in the urine, which otherwise follows the administration of methanol, was completely inhibited during repetitive administration of ethanol. Formate appeared 1 to 2 hr after the cessation of ethanol administration.

673. Kessler, A.
DER GEMEINSAME EINFLUSS VON ARZNEIMITTELN UND ALKOHOLGRENZKONZENTRATIONEN AUF DIE VERKEHRSSICHERHEIT AN HAND DER ÜBERPRÜFUNG VON 2500 FÄLLEN. [The combined influence of drugs and alcohol on traffic safety; survey of 2,500 cases].
 Dissertation, Medical Faculty of the University of Mainz, West Germany, 29 pp. (35 ref.), 1963.
 G – stat. surv. – DC (add., infra-add., unspec. incr.) – mot. vehic. – blood lev. – mot. perform. – psychol. perform. – analg., antipyret. – sed., hypnot. – *CAAAL-0 A-0051.
- A survey was conducted on 2500 traffic accident cases in which use of alcohol was established. The blood alcohol level in these cases was 1.5°/oo or more. 99% were male, 1% female. 10.3% admitted that they had taken drugs within the last 24 hr. More than half of the drugs were analgesics and antipyretics, followed by sedatives and hypnotics. 8 cases are discussed in detail. In most of the latter cases, alcohol-drug synergism was clearly established. It is concluded that drivers should be warned more effectively about the dangers of simultaneous alcohol and drug ingestion.
674. Kettenmeyer, G.
L'INFLUENCE DE CERTAINS MÉDICAMENTS DANS L'INTOXICATION ALCOOLIQUE (ANTAGONISMES ET SYNERGIES). [Influence of certain drugs on alcoholic intoxication (antagonisms and synergisms)].
 Ann. Med. Leg. (Paris), 28: 75-80 (40 ref.), 1948.
 F – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – mammals – blood lev. – CNS – respir. – amphetamines – analg., antipyret. – autonomic agents – barbiturates – hormones, hormone antag. – stimulants – *CAAAL-4960-A15 A-0830.
- Reviewed is the literature on substances which might influence the degree of alcohol intoxication, both biochemically and clinically. To date, the results of most experiments are contradictory and much more research is needed. Research is discussed concerning the following drugs: caffeine, benzedrine, pervitin, ephedrine, adrenalin, metrazol, strychnine, barbiturates, morphine, thyroxine, and dinitro-derivatives.
675. Kew, M. C., Bersohn, I., and Siew, S.
POSSIBLE HEPATOTOXICITY OF CANNABIS.
 Lancet (London), 1(7594): 578-579 (5 ref.), 1969.
 E – general – DC (unspec.) – humans – blood comp., sites, lymph – liver, kidney – hallucinogens – *CAAAL-0 B-0338.
- The authors wondered if, since the active constituents of marihuana are known to be concentrated in the liver, its long-term use might cause liver damage, or perhaps potentiate the toxic effects of alcohol on the liver. Biochemical and histological changes found in 12 marihuana smokers were significant but not conclusive. The authors also noted that alcohol consumption was unusual in the subjects studied, who claimed that alcohol interfered with the "high" normally achieved with marihuana.
676. Khan, A. U., Forney, R. B., and Hughes, F. W.
EFFECT OF TRANQUILIZERS ON THE METABOLISM OF ETHANOL.
 Arch. Int. Pharmacodyn. (Gand), 150(1-2): 171-176 (15 ref.), 1964.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – mammals – acute admin. – in vivo – blood lev. – other drug lev. – species or sex diff. – CNS – metab. proc. – tranquilizers – *CAAAL-0 A-0831.

Male mice were given 4 g/kg ethanol ip, plus 20 mg/kg benzquinamide ip or saline. Also, 28 human subjects were given 2 oz absolute ethanol/kg and 25 mg benzquinamide; a control group received placebo. In a third experiment, male albino rats were administered 2 g/kg ethanol ip, plus saline or 1 of the following drugs ip (administered simultaneously with ethanol, except for reserpine which was given 1 hr prior to ethanol): 20 mg/kg chlordiazepoxide, 20 mg/kg benzquinamide, 10 mg/kg hydroxyzine, 5 mg/kg chlorpromazine, 0.5 mg/kg reserpine, and 100 mg/kg meprobamate. It was found that the rate of disappearance of ethanol was significantly decreased in the mice which received benzquinamide, but no change in the rate was noted in humans. None of the 6 tranquilizers produced any change in the blood ethanol levels in rats, determined 1 hr after ethanol.

677. Khanna, J. M., and Kalant, H.
EFFECT OF INHIBITORS AND INDUCERS OF DRUG METABOLISM ON ETHANOL METABOLISM *IN VIVO*.
Biochem. Pharmacol. (New York), 19(6): 2033-2041 (21 ref.), 1970.
E – exp. cont. – cross-tol. – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – blood lev. – absorp., distrib., stor. – CNS – liver, kidney – metab. proc. – autocoids – barbiturates – *CAAAL-0 B-0531.

The effects of inhibitors (SKF 525-A) and inducers (chlorcyclizine, phenobarbital) of the hepatic microsomal drug-metabolizing systems (MDMS) on ethanol metabolism were studied in rats and mice. It was found that ip administration of 50 mg/kg of SKF 525-A, 45 min before a test dose of ethanol (2 g/kg), did not affect the shape of the ethanol disappearance curve, but did appear to delay the absorption or distribution of ethanol. Chronic pretreatment with chlorcyclizine hydrochloride or phenobarbital sodium (50 and 100 mg/kg/day, respectively) had no significant effect (except a delayed onset of sleep in the phenobarbital group) on the onset and duration of sleep, the rate of fall of the blood ethanol concentration, or the calculated rate of ethanol metabolism produced by a test dose of ethanol (2 g/kg) given 24 hr after the last dose of either drug. It is concluded that the hepatic MDMS are probably not involved in ethanol metabolism in vivo, or in cross-tolerance between ethanol and other drugs.

678. Khouw, L. B., Burbridge, T. N., and Simon, A.
THE INHIBITION OF ALCOHOL DEHYDROGENASE BY CHLORPROMAZINE.
Fed. Proc. (Bethesda), 19: 280 (2 ref.), 1960.
E – abst. – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – in vitro – blood lev. – metab. proc. – sed., hypnot. – tranquilizers – *CAAAL-0 A-1294.

The inhibition of alcohol dehydrogenase by chlorpromazine was studied in vitro. Another study (Tipton, Dale L. et al, Amer. J. Physiol., 200: 1007-1010, 1961) which investigated the observation, made during a metabolic study of problem drinkers, that chlorpromazine increased the blood alcohol level, revealed that in rabbits the inhibition of the conversion of ethanol to acetaldehyde is the sole source of the elevated blood alcohol level. In this study, horse liver alcohol dehydrogenase (ADH) was isolated using a slight modification of the Bonnichsen and Brink method, and a measurement of its activity was made by determining the reduction of DPN to DPNH spectrophotometrically. The Michaelis constant of the isolated ADH compared favorably with previously determined values. It was found that the horse liver ADH was strongly inhibited by the addition of chlorpromazine at a concentration of 2.8×10^{-6} M (1 µg/ml). At a similar concentration, promethazine and promazine also inhibited the horse liver ADH, but not to the same degree.

679. Khouw, L. B., Burbridge, T. N., and Sutherland, V. C.
THE INHIBITION OF ALCOHOL DEHYDROGENASE. I: KINETIC STUDIES.
Biochim. Biophys. Acta (Amsterdam), 73: 173-185 (31 ref.), 1963.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – acute admin. – in vitro – dose resp. – metab. proc. – tranquilizers – *CAAAL-10751-B3 A-1293.

The inhibition of alcohol dehydrogenase by chlorpromazine (CPZ) was studied in preparations of horse-liver, rabbit-liver, and yeast alcohol dehydrogenase (ADH). The analytical methods of Dixon and of Lineweaver and Burk were used in determining the enzyme kinetics of the ADH enzyme system in the presence of various concentrations of CPZ. CPZ was demonstrated to be a potent inhibitor of horse-liver and rabbit-liver ADH, and only a slight inhibitor of yeast ADH. The inhibition was characterized as being instantaneous, partial in nature, and reversible. Neither the sulfhydryl groups or the zinc in the ADH molecule were found to be affected by the drug, which fact indicates the non-specific nature of the ADH-CPZ interaction. The inhibition, with respect to the alcohol concentration, was found to be non-competitive. The discrepancy between the concentration of inhibitor causing 50% inhibition (2.8×10^{-5} M) and the apparent K_i (1.7×10^{-5} M) is attributed to the very complex nature of the inhibition.

680. Kibrick, E., and Smart, R. G.
PSYCHOTROPIC DRUG USE AND DRIVING RISK: A REVIEW AND ANALYSIS.
 Journal of Safety Research (Chicago), 2(2): 73-85 (58 ref.), 1970.
 E – SEC – review – DC (add., infra-add., unspec. incr.) – DC (unspec.) – med.-leg. – mot. vehic. – humans – CNS – analg., antipyret. – antidepressants – barbiturates – sed., hypnot. – stimulants – tranquilizers – *CAAAL-14632 B-1053.

The incidence of drug use by drivers is assessed by reference to studies of: general populations, non-fatal vehicular accidents, and fatal vehicular accidents. Investigations have varied in terms of drugs studied, reliability of data collection procedures, and criteria for choosing sample populations. Few propositions have been clearly established, and no studies have been replicated. The studies cited do show that 35-50% of the general population run the risk of driving after drug use at least once/yr, that 7% of these persons are exposed to risks of drinking and driving while on psychotropes, and that 11-15% of accident drivers have taken a psychotropic drug prior to their accident. The veracity of drivers' statements about drug use is very low, and estimates derived from questioning are probably very conservative. Psychotropic drug use is most likely to be found among certain drinking driver groups, especially the fatally injured; the psychotropes thus do not represent a substitute for alcohol, but an additional element to be combined with each other or with alcohol. Barbiturates are the most commonly-found drugs among accident and non-accident drivers; however, amphetamine use data is inadequate, due to the lack of laboratory analyses in studies to date. A need for further studies on the association of psychotropic drug use with driving errors is clearly indicated.

681. Kielholz, P., Goldberg, L., Im Obersteg, J., Pöldinger, W., Ramseyer, A., and Schmid, P.
STRASSENVERKEHR, TRANQUILIZER UND ALKOHOL. [Road traffic, tranquilizers and alcohol].
 Deutsch. Med. Wschr. (Stuttgart), 92(35): 1525-1531 (10 ref.), 1967.
 G – exp. cont. – exp. comp. – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – psychol. perform. – mot. perform. – tranquilizers – *CAAAL-0 B-0339.

The question whether impaired driving may be due, directly or indirectly, to tranquilizers (chlor-diazepoxide and meprobamate) was investigated within the framework of a possible interaction with alcohol. 120 policemen were given placebo, 400 mg meprobamate, or 10 mg chlordiazepoxide, under double-blind conditions, with and without the addition of alcohol (sufficient white wine to produce a blood alcohol level of 0.8-1.0‰), to test their driving ability under natural conditions at a trial area. At the same time, determinations were made of the blood alcohol level, drug levels in the blood, reaction time, and subjective self-evaluation. The statistical evaluation of the results showed no direct effect of the applied drugs in the given dose on the blood alcohol level or on the alcohol effects. Conversely, impaired driving at an average blood alcohol value of 0.8‰ was established with high statistical significance.

682. Kielholz, P., Goldberg, L., Im Obersteg, J., Pöldinger, W., Ramseyer, A., and Schmid, P. CIRCULATION ROUTIÈRE, TRANQUILLISANTS ET ALCOOL. [Road traffic, tranquilizers and alcohol]. Hyg. Ment. (Paris), 56(2): 39-60 (10 ref.), 1967.
F – exp. cont. – exp. comp. – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – tranquilizers – *CAAAL-0 B-0340.

The question whether impaired driving may be due, directly or indirectly, to tranquilizers was investigated within the framework of a possible interaction with alcohol. 120 policemen were given placebo, 400 mg meprobamate, or 10 mg chlordiazepoxide, under double-blind conditions, with and without the addition of alcohol (sufficient white wine to produce a blood alcohol level of 0.8-1.0°/oo), to test their driving ability under natural conditions at a trial area. At the same time, determinations were made on the blood alcohol level, drug levels in the blood, reaction time, and subjective self-evaluation. The statistical evaluation of the results showed no direct effect of the applied drugs in the given dose on the blood alcohol level or on the alcohol effects; conversely, impaired driving at an average blood alcohol value of 0.8°/oo was established with high statistical significance.

683. Kielholz, P., and Pöldinger, W. PHARMAKA, DROGENABHÄNGIGKEIT UND VERKEHR. [Drugs, drug dependence, and traffic]. Schweiz. Med. Wschr. (Basel), 97(1): 1-8, and 97(2): 49-54 (55 ref.), 1967.
G – ES – general – review – DC (add., infra-add., unspec. incr.) – DC (sensit.) – mot. vehic. – humans – mammals – blood lev. – cardiovasc. – CNS – antidepressants – anti-infectants – sed., hypnot. – tranquilizers – *CAAAL-0 B-0341.

The authors review in detail the various drug groups which may directly or indirectly impair driving ability. Various pharmaceutical agents, especially those with CNS-depressant qualities (e.g., hypnotics, sedatives, neuroleptics, tranquilizers, antidepressants, and antihistamines) may enhance the action of alcohol, and other agents (animal charcoal, chloroquine, furazolidone, isoniazid, metronidazole, sulfonyleureas, calcium cyanide, n-butyraldoxime, thiocyanate compounds, and *Coprinus atramentarius*) may result in antabuse-like reactions. Great importance must be attributed to the fact that the alcohol-enhancing action and side effects of both neuroleptics and antidepressants gradually disappear following long-term administration; thus, the greatest risk occurs after single doses, and the hazard decreases with continued therapy.

684. Kielholz, P., Goldberg, L., Im Obersteg, J., Pöldinger, W., Ramseyer, A., and Schmid, P. FAHRVERSUCHE ZUR FRAGE DER BEEINTRÄCHTIGUNG DER VERKEHRSTÜCHTIGKEIT DURCH ALKOHOL, TRANQUILIZER UND HYPNOTIKA. [Tests of driving ability for estimating the effects of alcohol, tranquilizers and hypnotics]. Deutsch. Med. Wschr. (Stuttgart), 94(7): 301-306 (8 ref.), 1969.
G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – barbiturates – sed., hypnot. – stimulants – *CAAAL-0 B-0342.

200 healthy subjects of different age groups were evaluated for driving ability and reaction time, before and after the intake of (a) placebo, (b) 20 mg chlordiazepoxide, (c) 800 mg meprobamate, (d) 200 mg phenobarbital, or (2) 200 mg methyprylone, alone and in combination with alcohol. The content of alcohol and drug in the blood was determined quantitatively. The results revealed that a single dose of (b) or (c) increased somewhat the errors made during the driving test. The intake of (d) or (e) increased the errors to a statistically more significant extent. The combination of (b), (c), (d) or (e) with alcohol had a synergistic effect. Blood alcohol levels of 0.8°/oo significantly increased the errors made during driving (probability less than 0.001).

685. Kielholz, P.

FAHRVERSUCHE ZUR FRAGE DER BEEINTRÄCHTIGUNG DER VERKEHRSTÜCHTIGKEIT DURCH ALKOHOL, TRANQUILIZER UND HYPNOTIKA.

[Studies on the question of impairment of driving ability by alcohol, tranquilizers, and hypnotics].

In: *Alkohol und Verkehrssicherheit*: Konferenzbericht der 5. Internationalen Konferenz über Alkohol und Verkehrssicherheit. [Alcohol and traffic safety: proceedings of the 5th International Conference on Alcohol and Traffic Safety.] Freiburg im Breisgau, West Germany, 1969. Freiburg im Breisgau: Hans Ferdinand Schulz Verlag, pp. III.53-III.54 (0 ref.), 1969.

G – exp. cont. – exp. comp. – presentation – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – CNS – sed., hypnot. – tranquilizers – *CAAAL-0 B-0946.

An experiment is reported as an introduction to a film presentation. The effects, on driving ability, of frequently-prescribed tranquilizers and hypnotics, alone and in combination with alcohol, were investigated in 320 volunteers from the Basel police force. The subjects received po, under double blind conditions, a placebo, 10 or 20 mg chlordiazepoxide, 400 or 800 mg meprobamate, 200 mg phenobarbital, or 200 mg methyprylone. In addition, half of the subjects also received sufficient alcohol to achieve a blood alcohol concentration of 0.8°/oo 1 hr after ingestion. Statistical evaluation of the results showed that lower doses of the tranquilizers did not significantly influence driving ability or the effects of alcohol; larger doses, while not affecting driving ability, did significantly enhance the effects of alcohol. Both hypnotics led to a decline in driving performance, and both enhanced the effect of alcohol. Also presented as a paper in *Alkohol und Verkehrssicherheit*, pp. III. 54a-III.54b, 1969.

686. Kielholz, P.

ALKOHOL, MEDIKAMENTE UND VERKEHR. [Alcohol, drugs, and traffic].

In: *Alkohol und Verkehrssicherheit* Konferenzbericht der 5. Internationalen Konferenz über Alkohol und Verkehrssicherheit. [Alcohol and traffic safety: proceedings of the 5th International Conference on Alcohol and Traffic Safety.] Freiburg im Breisgau, West Germany, 1969. Freiburg im Breisgau: Hans Ferdinand Schulz Verlag, pp. III.54a-III.54b (2 ref.) @ 1969.

G – exp. cont. – exp. comp. – presentation – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – CNS – sed., hypnot. – tranquilizers – *CAAAL-0 B-0947.

The effects, on driving ability, of frequently-prescribed tranquilizers and hypnotics, alone and in combination with alcohol, were investigated in 320 volunteers from the Basel police force. The subjects received po, under double blind conditions, a placebo, 10 or 20 mg chlordiazepoxide, 400 or 800 mg meprobamate, 200 mg phenobarbital, or 200 mg methyprylone. In addition, half of the subjects also received sufficient alcohol to achieve a blood alcohol concentration of 0.8°/oo 1 hr after ingestion. Statistical evaluation of the results showed that lower doses of the tranquilizers did not significantly influence driving ability or the effects of alcohol; larger doses, while not affecting driving ability, did significantly enhance the effects of alcohol. Both hypnotics led to a decline in driving performance, and both enhanced the effect of alcohol. Also reported as an introduction to a film presentation in *Alkohol und Verkehrssicherheit*, pp. III.53-III.54, 1969.

687. Kiessling, K. -H., and Pilström, L.

EFFECT OF ETHANOL ON RAT LIVER. V. MORPHOLOGICAL AND FUNCTIONAL CHANGES AFTER PROLONGED CONSUMPTION OF VARIOUS ALCOHOLIC BEVERAGES.

Quart. J. Stud. Alcohol (New Haven), 29(4A): 819-827 (14 ref.), 1968.

E – exp. cont. – exp. comp. – congen. stud. – mammals – chronic admin. – in vivo – liver, kidney – metab. proc. – respir. – *CAAAL-0 B-0343.

Male rats were given solid food and 95% ethanol, cognac, or whiskey (all diluted to 15% v/v), vermouht (15% v/v), white wine (12% v/v), red wine (12% v/v), or water and isocaloric glucose

for 9 months. Liver biopsies at 150 days showed a significant increase in mitochondrial size, except for white wine drinkers. At 270 days, enlargement was pronounced in the white wine group, but a striking return to control values had occurred in all other groups except those given ethanol. The results suggest that congeners present in the beverages strengthened the effects of the ethanol on certain of the functions studied, and counteracted its effect on others.

688. Kieve, R.

CLINICAL MANAGEMENT OF ACUTE INTOXICATION IN THE CHRONIC ALCOHOLIC.

American Practitioner and Digest of Treatment (Philadelphia), 1(7): 743-746 (4 ref.), 1950.
E – general – DC (unspec.) – drug-dep. humans – cardiovasc. – CNS – glands – liver, kidney – metab. proc. – respir. – analg., antipyret. – barbiturates – nutritive agents – sed., hypnot. – *CAAAL-5579-N1 A-0832.

Treatment of acute intoxication includes the following routine measures: adequate sedation, oxygen to accelerate blood alcohol oxidation, fluids to flush the metabolites out of the system, and an adequate supply of B-complex vitamins and 500 mg ascorbic acid. The sedative of choice is paraldehyde, which has a wide margin of safety. Sedation is commonly initiated with 12 cc paraldehyde po, 0.2 g pentobarbital sodium by rectum, and 50 mg demerol hypodermically. The pentobarbital sodium and demerol are not repeated. It must always be kept in mind that the acutely-intoxicated but not stuporous alcoholic needs a larger amount of paraldehyde than the normal patient. Also, 1/4 g myanesin, 3-6 times/day, effectively decreases or abolishes tremors in the acutely-intoxicated person, in either the presence or absence of delirium tremens.

689. Kirchheim, D.

TOXIZITÄT UND WIRKUNG EINIGER THIURAMDISULFIDVERBINDUNGEN AUF DEN ALKOHOLSTOFFWECHSEL. [Toxicity and effect of a few thiuramdisulfide compounds on alcohol metabolism].

Naunyn Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 214: 59-66 (47 ref.), 1951.
G – exp. cont. – exp. comp. – DC (sensit.) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – miscellaneous – *CAAAL-5929-A2 A-1336.

It has been observed that thiuramdisulfide compounds induce intolerance towards alcohol. Since antabuse (tetraethylthiuram disulfide) (TA) was known to have these effects, a series of experiments were performed to study the effect of other thiuramdisulfide compounds, namely tetramethyl-(TM), dipiperidyl-(DP), dimethyl-diphenyl-(DM), and diethyl-diphenyl-thiuramdisulfide (DA). Since all 5 compounds were insoluble in water, an emulgator was used. The toxicities of the individual compounds were determined in white mice, using po and intrastomacal routes, and the results were tabulated. Tests were then carried out on rabbits to study the effect of the compounds on the alcohol metabolism. The animals received 30 cc of 10% alcohol on the first day, and the blood acetaldehyde concentrations were determined by the Stolz method. On second day, the animals received a dose of thiuramdisulfide compound, followed by another blood test within 14 to 18 hr. The results were again tabulated. DM, DA, and DP did not seem to have antabuse-like effects; however, both TM and TA did influence the acetaldehyde level in the blood. The author concludes that a connection exists between the chemical constitution of the thiuramdisulfide compounds and their ability to increase the acetaldehyde level in the blood, when administered in combination with alcohol.

690. Kitto, W.

ANTIBIOTICS AND INGESTION OF ALCOHOL.

J.A.M.A. (Chicago), 193(5): 411 (0 ref.), 1965.
E – general – DC (decrease) – humans – absorp., distrib., stor. – G.I. tract – anti-infectants – *CAAAL-11238-B3 B-0344.

In answer to the question whether alcohol interferes with antibiotics in man, it is stated that the degradation of all penicillin is enhanced by the action of alcohol. The time of inactivation varies with the alcohol concentration, the penicillin concentration, and the particular penicillin molecule involved. If 3 hr are allowed to elapse after ingestion of 30 cc alcohol, there should be too little alcohol left in the gastrointestinal tract to interfere with the stability of the penicillin molecule; in fact, moderate inflammation of the intestine by alcohol may enhance absorption—Kanamycin sulfate can be absorbed in appreciable quantities if taken as an alcoholic elixir rather than a dry powder. If penicillin is ingested first, the effect of alcohol should be negligible after 2 hr.

691. Kjølstad, T.

MEDIKAMENTELL BEHANDLING AV AKUTT ALKOHOLRUS OG BAKRUS. [Drug therapy of acute alcohol intoxication and hangover].

T. Norsk. Laegeforen. (Oslo), 87: 1284-1285 (0 ref.),

1967.

N – general – DC (unspec.) – drug-dep. humans – psychol. perform. – absorp., distrib., stor. – CNS – G.I. tract – elect., water-bal. agents – sed., hypnot. – tranquilizers – *CAAAL-0 B-0345.

As preparation for subsequent socio- and psychotherapy, the author recommends 200-300 mg chlorpromazine initially, tapering off to 50 mg, plus 25-50 mg promethazine in the evening. Barbiturates, chloral hydrate, and paraldehyde should be avoided. Disulfiram, 0.2-0.4 g/day, is an aid to abstinence, but not a therapy in itself. In cases of severe intoxication, an emetic or stomach pump may be used to prevent further alcohol absorption; 100 mg chlorpromazine or 50 mg promethazine are effective in inducing sleep. For hangovers, 5 g of sodium chloride plus 1/4 l of liquid and 50 mg promethazine should bring relief within 20 min, and 100 mg chlorpromazine may be used if necessary.

692. Klaasen, C. D.

ETHANOL METABOLISM IN RATS AFTER MICROSOMAL METABOLIZING ENZYME INDUCTION.

Proc. Soc. Exp. Biol. Med. (New York), 132: 1099-1102 (12 ref.),

1969.

E – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – barbiturates – *CAAAL-0 B-0948.

The effects of pretreatment with agents known to stimulate drug metabolism, on the rate of disappearance of ethanol from the blood of rats were studied. Phenobarbital sodium (75 mg/kg), gamma-chlordane (50 mg/kg), chlorcyclizine hydrochloride (25 mg/kg), and 3-methylcholanthrene (20 mg/kg) were given ip daily for 3 days. 3 g/kg ethanol were given by gavage 24 hr after the last pretreatment dose, and blood concentrations were measured hourly for 8 hr. Zoxazolamine paralysis time, based on righting reflex, was used as the measure of stimulation of microsomal enzymes. All drugs studied decreased zoxazolamine time 1/2 to 20-fold, indicating an increased drug metabolism. Peak concentrations of ethanol occurred 2 hr after administration, and disappeared at a linear rate from 4-8 hr. Phenobarbital and 3-methylcholanthrene did not alter ethanol concentrations at any time, while chlorcyclizine tended to increase concentration, and differences were significant at the 3 and 4 hr collection intervals. Chlordane also tended to increase blood ethanol, but the difference was significant only at the 3 hr interval. As no significant change in the rate of ethanol disappearance was found, it is concluded that agents which increase the microsomal metabolism of drugs may have little quantitative importance in determining the rate of ethanol metabolism in the rat.

693. Klein, H.

ALKOHOL UND MEDIKAMENTE. I. DURCH MEDIKAMENTE VERURSACHTE ALKOHOLUNVERTRÄGLICHKEIT UND VERSTÄRKTE ALKOHOLWIRKUNG.

[Alcohol and drugs. I. Loss of tolerance to alcohol and potentiation of the effects of alcohol caused by drugs].

Fortschritte der Medizin (Berlin), 82(5): 169-172 (1 ref.),

1964.

G – review – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – blood lev. – respir. – anti-infectants – barbiturates – sed., hypnot. – *CAAAL-0 A-0833.

The literature on alcohol sensitization or enhancement by drugs is reviewed. Sensitization reactions are discussed with respect to *Coprinus atramentarius*, calcium cyanamide, antabuse, animal charcoal, furoxone, sulfonylureas, irgapyrin, isonicotinic acid hydrazide, resochin, and rhodanates. Synergism is discussed with reference to barbiturates, isonicotinic acid hydrazide, pyrazine, streptomycin, and paraldehyde. 2 cases are mentioned in which patients, admitted to hospital to be treated for minor injuries, and with blood alcohol levels of 1.57°/oo and 1.60°/oo, were given eunarcon and trapanal, respectively. Both died within 1 1/2 hr, from edema of the lungs.

694. Klein, H.

ALKOHOL UND MEDIKAMENTE. II. DURCH MEDIKAMENTE VERÄNDERTE ALKOHOLWIRKUNG. [Alcohol and drugs. II. Change in alcohol effects caused by drugs].

Fortschritte der Medizin (Berlin), 82(9): 335-337 (43 ref.), 1964.

G – exp. cont. – exp. comp. – review – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – mot. perform. – CNS – autonomic agents – hallucinogens – *CAAAL-0 A-0834.

2 groups of human subjects drank 750 ml wine within a short time. 1 group received a tablet containing 15 mg ephedrine and 0.1 mg atropine, the other group a placebo. Two subjects are described in detail as typical for the whole group. Subject A. 1) under placebo and alcohol, subject is in good mood, sings, talks very much. Appears to be sober again after 5 hr. 2) under drug and alcohol, becomes intoxicated after 1 hr, starts to turn over chairs, and becomes very hostile after 2 hr. After 5 hr, still intoxicated, but calms down. Subject B. 1) similar to A, but gets tired after 3 hr, and appears sober after 5 hr. 2) sings and shouts after 1 hr, becomes very agitated and lascivious after 2 hr, speaks with difficulty, and, after 3 hr, complains about thirst. He calms down during the next 2 hr, and is tired, thirsty, and bad-tempered after 5 hr. The effect of a large number of psychotropic drugs on the action of alcohol is discussed. Statistics showed that, of 1500 impaired drivers, 137 had taken both alcohol and drugs. Of this group, the most frequently used drugs were: neurophilin, gelonida antineuralgic tablets, optalidon, librium, and katovit; those drugs to be regarded as having an effect on the alcohol intoxication were dolviran, irgapyrin, eusedon, and optalidon, and those which had a possible effect were neurophylin, librium, and katovit.

695. Klein, H.

ALKOHOL UND MEDIKAMENTE. III. DIE BESTIMMUNG DER UNVERTRÄGLICHKEIT. [Alcohol and drugs. III. The determination of intolerance].

Fortschritte der Medizin (Berlin), 83(24): 977-980 (12 ref.), 1965.

G – general – DC (sensit.) – humans – mammals – blood lev. – cardiovasc. – anti-infectants – elect., water-bal. agents – hormones, hormone antag. – miscellaneous – unclass. ther. agents. – *CAAAL-0 B-0346.

The author discusses the following with reference to alcohol intolerance; salyrgan, antabuse, tetramethylthiuram monosulfide, tetramethylthiuram disulfide, chlorpropamide, butyraldoxime, furazolidone, 1,10-phenanthroline, 8-hydroxyquinoline, benzthiazide, dihydrobenzthiazide, chlorothiazide, dihydrochlorothiazide, 2-amino-5-nitrothiazole, 2-(2,2,2-trifluoroacetamide)-5-nitrothiazole, and 2-thiazolidine-ethion. Several tests for the determination of possible alcohol-drug intolerance reactions are proposed. Firstly, the inhibition by the substance of the nicotinamide-adenine dinucleotide-dependent aldehyde dehydrogenase should be determined. Then, the following should be established: a) blood pressure, pulse frequency, and skin temperature, b) calcium concentration of the blood, and c) acetaldehyde and pyruvic acid concentrations during alcohol ingestion. Since quite a few new drugs have shown this incompatibility with alcohol, it is suggested that this effect should be investigated before new drugs are put on the market.

696. Klein, H. A.
 THE USE OF ALCOHOL IN TREATMENT OF CARBOLIC ACID BURNS AND POISONING.
 J.A.M.A. (Chicago), 35: 1557 (0 ref.), 1900.
 E – general – case hist. – DC (antidotal) – humans – G.I. tract – skel., muscle, skin – anesthetics
 – *CAAAL-0 A-0835.

The author relates his experiences with alcohol as an antidote for carbolic acid by citing 3 case histories. In the first case, carbolic acid had burned the face and hands; the affected parts were washed with alcohol 15 min later, with immediate improvement. In the second case, 4 oz doses of alcohol were given every 1/2 hr, followed by 1 oz every hr, to a woman who had taken 2 oz carbolic acid 10 min prior to receiving medical attention. In the last case, an infected arm was treated with 4 applications of 95% carbolic acid, followed 1 min later by alcohol. In every case there was complete recovery.

697. Kliewe, H.
 ÜBER UNTERSCHIEDE VERSCHIEDENER WEINSORTEN IN MEDIZINISCHER SICHT. [The differences in various kinds of wine from the medical point of view].
 Wein-Wissenschaft (Wiesbaden), 16(11): 177-196 (0 ref.), 1961.
 G – exp. comp. – congen. stud. – mammals – acute admin. – in vivo – dose resp. – blood lev. – other drug lev. – blood comp., sites, lymph – G.I. tract – glands – liver, kidney – metab. proc. – *CAAAL-0 A-1409.

The following aspects were investigated: a) the effects of ethanol + allyl alcohol or white wine + allyl alcohol on the liver and serum glutamate-pyruvate-transaminase (SGPT) activity of white rats, b) the effects of alcohol, wine, or sulphurous acid (SA), with or without pretreatment with suprarenin, glucose, or insulin, on the blood sugar levels of rabbits, c) the effect of po administration of wine or SA, or the injection of insulin or suprarenin, on the bactericidal power and properdin level of blood in rabbits, and d) the effect of po, sc, and intracardial administration of wine, ethanol, or SA, as well as other acids and potassium sulphate, on adrenal gland secretions of guinea pigs and rats. Both wine and ethanol enhanced allyl alcohol-induced liver damage, and both increased SGPT activity, wine more strongly than ethanol. Blood sugar levels were only increased by SA; wine increased the properdin level by 100% in 24 hr. Small and large wine doses, and small ethanol doses, increased adrenal secretion, while large ethanol doses decreased it. It is concluded that 1 bottle of wine/day, containing average amounts of ethanol and congeners, should be harmless to a healthy, well-nourished individual.

698. Kliewe, H., Gillissen, G., and Wiener, K.
 DIE WIRKUNG VON HYBRIDENWEIN AUF DIE LEBER. [The effect of hybrid wine on the liver].
 Experientia (Basel), 17: 42 (5 ref.), 1961.
 G – exp. cont. – congen. stud. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – liver, kidney – alcohols – *CAAAL-0 A-1410.

Experiments were performed on male rats (145-155 g), to study the effects on allyl alcohol-induced liver damage of "pure" wine (from grapes of pure European origin), hybrid wine (from grapes developed by a crossing of European and native American varieties), and ethanol sol. Rats received 2 ml po of a 12.79 vol% concentration of ethanol sol, pure wine, or hybrid wine, or a control administration of water, 1 hr prior to po administration of 0.6 ml 35% allyl alcohol. The rate of necrosis and serum proteins were studied. Significant increases of liver damage were caused by all test sol in the following, increasing, order of intensity: ethanol sol, pure wine, and hybrid wine. Electrophoretic measurements of serum proteins showed only a percentage change in concentration of the individual components between test groups and controls, indicating the influence of allyl

alcohol, but not any additional damage. Also reported, in expanded form, in *Medicina et Pharmacologia Experimentalis*, 4: 227-233, 1961.

699. Kliewe, H., Gillissen, G., and Nessling, W.
DIE SERUM-GLUTAMAT-PYRUVAT-TRANSAMINASE ALS INDIKATOR FÜR LEBERSCHÄDEN NACH GABEN AETHANOLHALTIGER FLÜSSIGKEITEN. [Serum glutamate pyruvate transaminase as indicator of liver damage after doses of liquids containing alcohol].
Medizin und Ernährung (Munich), 2(6): 127-129 (21 ref.), 1961.
 G – exp. cont. – exp. comp. – congen. stud. – mammals – acute admin. – in vivo – blood comp., sites, lymph – liver, kidney – *CAAAL-0 A-1411.

The comparative liver damage induced by ethanol “pure” white wine (made from grapes of pure European origin), and hybrid wine (made from grapes developed from a crossing of European and native American varieties), using serum glutamate-pyruvate-transaminase (SGPT) activity as criterion, was determined. Male rats (140-160 g) were pretreated with a single 1 ml dose of 1% allyl alcohol. Then, 2 ml of 12.8% ethanol, pure wine, red hybrid wine, or white hybrid wine, were given at 1 hr, and again at 12 hr. 30 hr after the beginning of the experiment, the animals were sacrificed, and the serum analyzed. The SGPT activity was increased, in ascending order, as follows: untreated controls, allyl alcohol, allyl alcohol plus ethanol, allyl alcohol plus pure white wine, allyl alcohol plus white hybrid wine, and allyl alcohol plus red hybrid wine. It is concluded that greater liver damage is liable to occur by ingestion of hybrid wines, or of European wines with a high content of volatile oils and other substances.

700. Kliewe, H., Gillissen, G., and Wiener, K.
VERGLEICHENDE UNTERSUCHUNGEN ÜBER BIOLOGISCHE WIRKUNGEN VON HYBRIDENWEIN. [Comparative investigations on the biological action of hybrid wine].
Med. Pharmacol. Exp. (Basel), 4: 227-233 (19 ref.), 1961.
 G – exp. cont. – congen. stud. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – liver, kidney – alcohols – *CAAAL-0 A-1412.

Experiments were performed on male rats (145-155 g), to study the comparative effects on allyl alcohol-induced liver damage of “pure” wine (from grapes of pure European origin), hybrid wine (from grapes developed by a crossing of European and native American varieties), and ethanol sol. Rats received 2 ml po of a 12.79 vol% concentration of ethanol sol, pure wine, or hybrid wine, or a control administration of water, 1 hr prior to po administration of 0.6 ml 35% allyl alcohol. The rate of necrosis and serum proteins were studied. Significant increases of liver damage were caused by all test sol in the following, increasing, order of intensity: ethanol sol, pure wine, and hybrid wine. Electrophoretic measurements of serum proteins showed only a percentage change in concentration of the individual components, between test groups and controls, indicating the influence of allyl alcohol, but not any additional damage. Possible ways of reducing biological errors in determining the rate of necrosis are discussed. Also reported, in condensed form, in *Experientia*, 17: 42, 1961.

701. Knick, B., and Wagner, H. -J.
THERAPEUTISCHE UNTERSUCHUNGEN MIT ACTH, CORTISON UND PREDNISOLON (SOLU-DECORTIN-H) BEI AKUTER ALKOHOLINTOXIKATION. (BEOBACHTUNGEN BEI DER BEHANDLUNG VON VOLLRAUSCHZUSTÄNDEN JUGENDLICHER, KLINISCH-EXPERIMENTELLE UND TIEREXPERIMENTELLE ALKOHOLBELASTUNGEN). [Therapeutic studies with ACTH, cortisone and prednisolone (solu-decortin-H) in acute alcohol intoxication (observations on the treatment of drunkenness in young persons, clinical and animal experiments with acute intoxication)].
Ärztliche Wochenschrift (Berlin), 13(5): 1110-1114 (32 ref.), 1958.

G – exp. comp. – DC (antidotal) – DC (decrease) – DC (unchanged) – humans – mammals – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – metab. proc. – elect., water-bal. agents – musculoskel. agents – *CAAAL-8901-A1 A-0836.

Prednisolone (100-150 mg) or cortisone (300 mg) were given to 17 young persons admitted in states of acute alcohol intoxication; those treated with prednisolone seemed to recover sooner than those who received cortisone or laevosan, i.e., the clinical condition improved but the alcohol metabolism was not affected. In experiments with dogs, these findings were confirmed.

702. Knott, D. H., Barlow, G., and Beard, J. D.
EFFECTS OF ALCOHOL INGESTION ON THE PRODUCTION OF AND RESPONSE TO EXPERIMENTAL HEMORRHAGIC STRESS.
New Eng. J. Med. (Boston), 269(6): 292-295 (6 ref.), 1963.
E – SEC – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – cardiovasc. – barbiturates – *CAAAL-10747-B2 A-1413.

Pentobarbital sodium was given iv as anesthetic 30 min before standardized hemorrhagic stress was applied to 10 experimental and 5 control dogs. Ethanol (3 g/kg) was given as a 30% sol via gastric tube. 75 min prior to hemorrhage by withdrawal of 200 cc blood from a femoral artery. All alcohol-treated animals showed every sign of intoxication. One of most striking differences observed between control and alcohol-treated dogs was an apparent reduction in blood plasma vol in the latter before hemorrhage; this was not accompanied by a decreased hematocrit. Unlike the alcohol-treated group, the control animals had a continuing increase in heart rate after hemorrhage. However, the percentages in blood loss, compensation, and blood-vol reduction, and the apparent hemodynamic response after hemorrhage, were essentially the same in both groups. It is concluded that the data fail to indicate any deleterious effect of ethanol on the response to moderate hemorrhagic stress in pentobarbital-anesthetized dogs.

703. Koe, B. K., and Tenen, S. S.
BLOCKADE OF ETHANOL METABOLISM AND REDUCED ALCOHOL SELECTION IN C57BL MICE BY BUTYRALDOXIME.
Fed. Proc. (Bethesda), 28(2): 546 (0 ref.), 1969.
E – abst. – exp. – DC (sensit.) – mammals – in vivo – in vitro – blood lev. – cardiovasc. – metab. proc. – miscellaneous – *CAAAL-0 B-0347.

N-butyraldohime, an antialcohol compound in man, caused a marked and prolonged reduction in ethanol selection by C57 BL mice, a strain with a natural preference for ethanol. In vitro butyraldohime is not a liver aldehyde dehydrogenase (CHO/DH) inhibitor, but is a potent alcohol dehydrogenase (OH/DH) inhibitor (IC_{50} 10^{-6} M competitive with ethanol, non-competitive with nicotinamide adenine dinucleotide). In vivo butyraldohime (1.5 mM/kg ip) reduced OH/DH activity by 75% in 1 hr; activity returned to normal by 17 hr after drug administration. Moreover, it decreased CHO/DH activity by 50-70% from 1 to 24 hr. After chronic administration of butyraldohime (in drinking fluid at 1 mg/ml), only CHO/DH activity was decreased, not OH/DH activity. Mice given chronic administration of butyraldohime in drinking fluid at 1 mg/ml, plus ethanol, showed a substantial level of ethanol, which probably accounts for the antialcohol property of butyraldohime.

704. Koe, B. K., and Tenen, S. S.
INHIBITING ACTION OF N-BUTYRALDOXIME ON ETHANOL METABOLISM AND ON NATURAL ETHANOL PREFERENCE OF C57BL MICE.
J. Pharmacol. Exp. Ther. (Baltimore), 174(3): 434-449 (40 ref.), 1970.
E – exp. cont. – DC (sensit.) – mammals – acute admin. – chronic admin. – in vivo – in vitro – dose resp. – blood lev. – blood comp., sites, lymph – liver, kidney – *CAAAL-0 B-0949.

Male mice and rats received butyraldoxime in drinking water or ip. The liver alcohol dehydrogenase (ADH) activity of mice was much reduced at 1 hr, but steadily increased to control values by 17 hr. Butyraldoxime exerted a negligible in vitro inhibition of beef or mouse liver aldehyde dehydrogenase (AldDH), yet there was a notable decrease of activity in livers of pretreated mice. Chronic intake of butyraldoxime and isobutyraldoxime caused a rise in hepatic ADH activity, and a reduction of AldDH activity. The latter returned to normal 1-2 days after withdrawal of the drug. After butyraldoxime ip, the normal rate of decline of blood ethanol levels induced by administration of 2-3 g/kg ethanol was markedly decreased in mice and rats. The rate of ethanol disappearance varied inversely with the interval between the 2 administrations. Similar effects were obtained in rats drinking butyraldoxime for 6 days, and then given 2 g/kg ethanol ip. Analogous effects on ethanol metabolism were found for several homologous aldoximes. Simultaneous consumption of oxime and ethanol induced a strong and protracted decrease in the natural preference of C57BL mice for ethanol. The data suggest that the anti-alcohol action of butyraldoxime is derived from a hepatic aldehyde dehydrogenase blockade, which interferes with the metabolism of acetaldehyde derived from ethanol.

705. Koelsch, F.
 UEBER NEUARTIGE GEWERBLICHE ERKRANKUNGEN IN
 KALKSTICKSTOFFBETRIEBEN. [A novel industrial illness in calcium cyanamide
 production].
 Munchen Med. Wschr. (Munich), 61(3): 1869-1870 (0 ref.), 1914.
 G – general – DC (sensit.) – humans – cardiovasc. – *CAAAL-0 A-1414.

The author describes the symptoms of an industrial cyanamide poisoning which occurs only after alcohol consumption. A reddish discolouration of the face and body appears after the first 1-2 swallows of alcohol. Although the temperature of the discoloured parts does not rise, a slight quiver can be observed over the whole body. Breathing quickens, accompanied by the occasional light cough. Both the heart beat and pulse quicken (100-130/min), while the blood pressure remains normal or decreases slightly. The reflexes and sensorium remain normal. Attacks usually last 1-2 hr, depending on the amount of alcohol consumed. The after-effects are general exhaustion and chills. The origin of the problem is thought to be exposure to chemicals containing cyanamide, and, to prevent such episodes, it is suggested that workers avoid the dust that forms in the production of calcium cyanamide, and that alcohol abstinence be maintained. Rest and the application of ice, as well as emetics or laxatives, are suggested as treatment.

706. Koff, R. S., Lui, S., Carter, E. A., and Isselbacher, K. J.
 PHENOBARBITAL INHIBITION OF THE ETHANOL-INDUCED FATTY LIVER.
 Clinical Research (New York), 17: 305 (0 ref.), 1969.
 E – abst. – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo –
 liver, kidney – metab. proc. – barbiturates – *CAAAL-0 B-0532.

Since administration of ethanol results in accumulation of triglycerides in the liver, the effect of phenobarbital on the fatty liver induced by a single dose of ethanol was studied. Female rats were pretreated with sodium phenobarbital (80 mg/kg) or saline daily for 4 days. Then 4.8 g/kg ethanol was given directly into the stomach. 16 hr later, the hepatic triglyceride concentrations were determined. The triglyceride levels of the animals pretreated with phenobarbital were much lower than in the controls. Electron microscopy revealed the effects on the rough endoplasmic reticulum (RER). The saline pretreatment plus alcohol had only a mild effect on the RER, and phenobarbital pretreatment alone affected only the smooth endoplasmic reticulum, but there was a striking increase in the RER of hepatic cells in animals given phenobarbital plus ethanol. The blood alcohol levels 1-3 hr after alcohol administration were much higher in the phenobarbital-pretreated than in the saline-pretreated group. These results indicate that phenobarbital inhibits the production of acute ethanol-induced fatty liver, possibly by altering the hepatic ethanol metabolism.

707. Koff, R. S., Carter, E. A., Lui, S., and Isselbacher, K. J.
 PREVENTION OF THE ETHANOL-INDUCED FATTY LIVER IN THE RAT BY
 PHENOBARBITAL.
 Gastroenterology (Baltimore), 59(1): 50-61 (32 ref.), 1970.
 E – exp. cont. – DC (decrease) – mammals – acute admin. – chronic admin. – in vivo – in vitro –
 blood lev. – liver, kidney – metab. proc. – anticonvulsants – barbiturates – sed., hypnot. –
 *CAAAL-15162 B-1013.

Female rats (175-200 g) were divided into 2 groups, 1 receiving 80 mg/kg sodium phenobarbital ip/day for 4 days, and the other receiving saline. 4-6 hr after the last injection, 1/2 of each group was given 4.8 g/kg 50% ethanol intragastrically; the remaining animals were given saline. 16 hr later, the animals were killed and their livers excised. Hepatic lipids, and blood ethanol, lactate, and phenobarbital levels were determined. Saline-pretreated rats given ethanol showed a 3-fold increase in hepatic triglycerides, while rats in the phenobarbital group given ethanol showed only a mild triglyceride increase. Phenobarbital inhibition of hepatic triglyceride accumulation was accompanied by alterations of the endoplasmic reticulum of the hepatocyte; blood ethanol was markedly elevated, while blood lactate was unaffected. The increased incorporation of fatty acid into triglyceride, found in microsomal preparations after in vivo ethanol administration, did not occur in phenobarbital-pretreated animals. Oxidation of ethanol by liver slices and supernatant fractions was diminished in phenobarbital-pretreated animals and when phenobarbital was added in vitro to slices and supernatant fractions of control animals. Ethanol oxidation by hepatic microsomes was similar in phenobarbital- and saline-pretreated rats, and was not affected by in vitro addition of phenobarbital. It is suggested that phenobarbital inhibits the alcohol-dehydrogenase-mediated oxidation of ethanol.

708. Kofman, O.
 EXPERIENCE WITH RESERPINE (SERPASIL) AND PERPHENAZINE (TRILAFON) IN
 ACUTE ALCOHOLIC INTOXICATIONS AND ALCOHOLIC PSYCHOSIS.
 Canad. Med. Ass. J. (Toronto), 79(12): 988-991 (10 ref.), 1958.
 E – FS – general – DC (unspec.) – drug-dep. humans – cardiovasc. – tranquilizers –
 *CAAAL-8679-N9 A-0837.

143 patients were treated for acute alcohol intoxication with reserpine (dosage: 2.5 mg reserpine im upon admission, the same dose in 1 hr if the patient was still restless, and a third dose in 6 hr; after this, 0.25 or 0.50 mg po 4 times/day). All those presenting no complications were free of significant symptoms in 24-48 hr. A series of 114 alcoholic patients in an acute state were treated with perphenazine. The most satisfactory dosage was 10 mg perphenazine im on admission, repeated in 1 hr, and thereafter 5 mg as necessary. Patients in general improved rapidly in a period of 12-24 hr. It is concluded that perphenazine im is the most effective, least complex agent of any drug employed by the authors in the treatment of acute alcoholic states.

709. Köhler, C.
 DIE PRÜFUNG DER WECHSELWIRKUNG ZWISCHEN ALKOHOL UND
 MEDIKAMENTEN MITTELS VARIANZANALYSE. [Investigation of the interaction between
 alcohol and drugs using the variance analysis].
 Blutalkohol (Hamburg), 4(6): 325-329 (0 ref.), 1967.
 G – exp. comp. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – CNS – anticonvulsants
 – sed., hypnot. – *CAAAL-0 B-0348.

The usefulness of the analysis of variance as a statistical tool was demonstrated, to determine the interaction between alcohol and dimethylsulfoxide, mogadon, revonal, and tegretol in mice. The mean lethal dose of alcohol and the drugs was used, and the rate of survival served as criterion. It was found that not only the single action of alcohol and drug have to be added, but that also a reciprocal action

increases the effect. The alcohol-drug combinations were therefore "supra-additive". The three drugs tested varied in effect when combined with alcohol.

710. Koivusalo, M.
STUDIES ON THE EFFECT OF OTHER ALCOHOLS ON THE METABOLISM OF METHANOL IN RAT LIVER HOMOGENATES.
 Acta Physiol. Scand. (Stockholm), 45: 102-108 (18 ref.), 1959.
 E – exp. cont. – exp. comp. – DC (decrease) – mammals – in vitro – liver, kidney – metab. proc. – alcohols – *CAAAL-9416-A2 A-1337.

In view of the known ability of ethanol to decrease the toxic effects of methanol, the effects of higher aliphatic alcohols on methanol metabolism were studied in vitro. Ethanol, n-propanol, iso-propanol, and n-butanol were incubated, in addition to methanol with rat liver homogenates, and the accumulation of formaldehyde was determined. It was found that 300 and 600 μ M ethanol clearly inhibited formaldehyde accumulation, whereas similar doses of the other alcohols were without effect. Controls yielded 102 μ g formaldehyde, while the amounts of formaldehyde corresponding to 300 and 600 μ M alcohol doses, respectively, were: ethanol—25 and 15 μ g, n-propanol—97 and 86 μ g, iso-propanol—97 and 101 μ g, and n-butanol—108 and 103 μ g. The author finds some difficulty in explaining the results, since, if alcohol dehydrogenase (and, possibly, an auxiliary system) is responsible for methanol oxidation, then n-propanol and n-butanol should have had inhibitory effects. Methanol oxidation by the catalase system would better explain the findings, but some inhibition by n-propanol might nevertheless be expected. On the other hand, in view of the oxidation mechanism of iso-propanol, its failure to inhibit formaldehyde is comprehensible.

711. Kolodny, A. L.
SIDE-EFFECTS PRODUCED BY ALCOHOL IN A PATIENT RECEIVING FURAZOLIDONE.
 Maryland Med. J. (Baltimore), 11: 248 (2 ref.), 1962.
 E – general – DC (sensit.) – humans – cardiovasc. – G.I. tract – respir. – anti-infectants – *CAAAL-10659-C3 A-1295.

A case is presented describing the adverse reaction manifested by administering alcohol to patients taking the nitrofurone compound furazolidone, used in the therapy of diarrhoea. A syndrome of excessive skin flushing and dyspnea occurs, which is similar to the alcohol-disulfiram reaction. A 36 yr-old male, bothered by recurrent diarrhoea for 2 yr, was instructed to take 100 mg furazolidone tablets 4 times daily. The patient drank 2 oz of brandy in an effort to relieve abdominal cramps after his third dose of medication. Severe wheezing and dyspnea, accompanied by intense facial flushing, occurred within 1 hr, and lasted about an hr, no treatment being given. The next day, the patient, being curious, and still taking his medication, consumed a martini cocktail, and again developed facial erythema and wheezing which lasted for 30 min. The mechanism of this reaction may be similar to that of disulfiram. Clinicians are well aware of a similar syndrome produced by furaltadone-alcohol, but not of one brought about by furazolidone-alcohol.

712. Konwaler, B. E., and Noyes, C. B., Jr.
CARBON TETRACHLORIDE POISONING: REPORT OF CASES.
 Calif. Med. (San Francisco), 61(1): 16-20 (11 ref.), 1944.
 E – general – DC (add., infra-add., unspec. incr.) – post-mort. – humans – cardiovasc. – liver, kidney – anti-infectants – *CAAAL-0 A-0838.

3 cases (1 fatal) illustrating varying degrees of severity of intoxication from carbon tetrachloride poisoning by inhalation are presented. The workers had been drinking heavily over a weekend, and, upon returning to work, were exposed to carbon tetrachloride fumes. Necropsy findings of the fatal

case are described and discussed. 3 other men who had not been drinking were exposed to the fumes but failed to develop symptoms. The author concludes that the alcohol previously consumed was the foremost in importance of the various factors which played a synergistic role in the poisonings. Mentioned is the unpublished experience of M.D. Willcutts (Captain, Marine Corps, U.S.N.) concerning 20 men exposed to carbon tetrachloride for 3-4 hr, of whom only those 3 who had consumed considerable quantities of liquor prior to exposure became poisoned. It is considered that the augmentation of carbon tetrachloride toxicity by alcohol is due to the combined deleterious effect of both substances on the liver.

713. Koopmann, H., and Kempfski, H.
 BEOBACHTUNGEN ÜBER DEN EINFLUSS VON KAFFEEGENUSS AUF DEN
 BLUTALKOHOLGEHALT UND ÜBER DEN FORENSISCHEN WERT DES
 URINALKOHOLGEHALTS. [Observations on the influence of coffee on blood alcohol and on
 the forensic value of urine alcohol].
 Munchen. Med. Wschr. (Munich), 84(20): 780-781 (1 ref.), 1937.
 G – exp. – DC (decrease) – med.-leg. – humans – acute admin. – in vivo – blood lev. – other drug
 lev. – stimulants – *CAAAL-978-U5 A-0839.

The influence of coffee (100 cc strong coffee) on blood alcohol (after ingestion of 100 cc cognac) and the forensic value of urine alcohol measurement were investigated in 5 male subjects. The authors conclude that: coffee has an influence on the degree of intoxication but not on the blood alcohol level, that the alcohol odour of expired air decreases temporarily after coffee, and that urine alcohol and blood alcohol are not parallel. Urine alcohol is at first considerably lower than blood alcohol, but is finally higher. The urine alcohol level alone, therefore, gives no correct indication of the degree of intoxication. For forensic purposes, the blood alcohol level of persons who also drink strong coffee should be considered differently, because such persons are less impaired.

714. Kopf, R.
 ZUR FRAGE DER ALKOHOLPOTENZIERUNG DURCH PHENOTHIAZINE.
 [Potentiation of the effects of alcohol by phenothiazines].
 Arch. Int. Pharmacodyn. (Gand), 110(1): 56-64 (10 ref.), 1957.
 G – ES – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
 – chronic admin. – dose resp. – CNS – tranquilizers – *CAAAL-8475-D2 A-0840.

Sleeping time in mice was measured after subhypnotic doses of ethanol, alone or in combination with chlorpromazine or pecazine (various routes of administration). It was found that 1.25 mg/kg chlorpromazine ip, followed by 2 g/kg ethanol iv, enhanced the hypnotic effect of alcohol; 20 mg/kg pecazine ip enhanced the alcohol effect to a lesser degree. When 1.25 mg/kg chlorpromazine ip was followed by 3 g/kg ethanol iv 1 1/2 hr later, sleeping time was prolonged 1,960%. Larger drug doses increased potentiation, and the effects lasted longer. Ip administration produced the greatest effect. Repeated administration of drugs and ethanol showed that, after 3 days, the effect of both drugs was diminished to a small fraction of the initial value. The risk of alcohol potentiation by the drugs is less with pecazine than with chlorpromazine—the duration of potentiation by 10 mg/kg chlorpromazine is about 30 hr, and that of pecazine about 6 hr.

715. Kopmann, E., and Hughes, F. W.
 POTENTIATING EFFECT OF ALCOHOL ON TRANQUILIZERS AND OTHER
 CENTRAL DEPRESSANTS.
 Arch. Gen. Psychiat. (Chicago), 1: 7-11 (10 ref.), 1959.
 E – exp. cont. – exp. comp. – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mammals
 – acute admin. – in vivo – CNS – barbiturates – tranquilizers – *CAAAL-8826-J2 A-0841.

Anxiety, discrimination, and inability to respond were measured before and after injections of the following drugs, alone or in combination with 3 g/kg ethanol: sodium glutamate (2 g/kg), chlorpromazine (2 mg/kg), reserpine (0.5 mg/kg), pentobarbital (10 mg/kg), meprobamate (100 mg/kg), and phenaglycodol (100 mg/kg). It was found that the effect of ethanol on anxiety was potentiatingly reduced by meprobamate, chlorpromazine, and pentobarbital. Loss of discrimination by ethanol was potentiated by meprobamate, chlorpromazine, phenaglycodol, and pentobarbital. Inability to respond, as induced by ethanol, was potentiated by meprobamate, phenaglycodol, and chlorpromazine. Chlorpromazine was shown to be a true potentiator of ethanol, in that anxiety and discrimination were inhibited by combination with ethanol, but not by the drug alone, and inability to respond was significantly enhanced. Reserpine, on the other hand, did not show any effect in combination with ethanol, other than simple addition.

716. Koppanyi, T.

THE EFFECT OF ARSENITE ON ALCOHOL INTOXICATION.

Anat. Rec. (Philadelphia), 94: 377-378 (3 ref.), 1946.
E – abst. – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – metab. proc. – miscellaneous – *CAAAL-4438-D2 A-0842.

12 rabbits received 4 cc of ethanol/kg sc. 6 of these received 5 mg/kg sodium arsenite 15 min before the alcohol—all 6 died. The 6 controls receiving alcohol alone recovered, on the average, within 4 hr. 6 rabbits received 25 mg/kg nembutal iv, 3 of them premedicated with the same dose of sodium arsenite as in the first experiment. All recovered. It is concluded that arsenite potentiates the toxicity of alcohol, and increases its duration of action; the effect is not nonspecific (due to additive toxic effects on the CNS), because arsenite did not prolong nembutal narcosis.

717. Koppanyi, T.

TREATMENT OF ACUTE ALCOHOLIC POISONING.

In: Himwich, Harold E., ed. *Alcoholism: Basic Aspects and Treatment*. Washington: American Association for the Advancement of Science, Pub. No. 47, pp. 93-113 (47 ref.), 1957.
E – exp. – general – DC (antidotal) – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – drug-dep. humans – mammals – acute admin. – in vivo – blood lev. – other drug lev. – CNS – respir. – skel., muscle, skin – barbiturates – sed., hypnot. – *CAAAL-0 A-0843.

The 2 kinds of acute alcoholic intoxication—inebriation or excitement, and alcoholic anesthesia or coma—are defined, and the treatment for each type reviewed and discussed. The customary treatment of inebriation with aliphatic depressants (chloral hydrate, paraldehyde, or barbiturates), or anti-anxiety agents such as chlorpromazine, is not without risk, due to the dangers of additive synergism or even potentiation. When the blood alcohol level is high, inebriation may be converted into anesthesia—the author witnessed the death of 1 intoxicated patient who was given 3 g sodium amobarbital iv. The results of an experiment on the effects of alcohol-chlorpromazine combinations on mice are tabulated (see Koppanyi, T. et al, *Quart. J. Stud. Alcohol* (New Haven), Suppl. 1: 24-36, 1961). The recommendations of Kieve, Rudolph (*American Practitioner and Digest of Treatment* (Philadelphia), 1(7): 743-746, 1950) are criticized. Treatment of coma and analeptics is also discussed.

718. Koppanyi, T., Canary, J. J., and Maengwyn-Davies, G. D.

PROBLEMS IN ACUTE ALCOHOL POISONING.

Quart. J. Stud. Alcohol (New Haven), Suppl. 1: 24-36 (28 ref.), 1961.
E – exp. cont. – general – DC (antidotal) – DC (decrease) – DC (add., infra-add., unspec. incr.) – drug-dep. humans – mammals – acute admin. – in vivo – blood lev. – acid-base, blood pH, elect. – cardiovasc. – CNS – metab. proc. – respir. – barbiturates – elect., water-bal. agents – sed., hypnot. – tranquilizers – *CAAAL-0 A-0844.

"Acute alcohol poisoning" is redefined and reevaluated. As for alcoholic intoxication, the risk involved in treating patients whose blood alcohol concentration exceeds 150 mg/100 ml by the routine use of hypnotics (e.g., paraldehyde, barbiturates) or tranquilizers (e.g. phenothiazines, chlordiazepoxide) is emphasized. These agents may have a synergistic or potentiative effect causing coma; the author has repeatedly seen instances of rapid conversion of inebriation to coma. The results of an experimental study showed that, of 17 mice given 0.05 mg 95% ethanol, none slept; of 15 mice given 5.0 mg chlorpromazine/kg, none slept; and of 19 mice given both substances simultaneously (above doses), 3 did not fall asleep, 13 slept for 175 min (average), and 3 died. Treatment of alcoholic coma and other acute manifestations in alcoholics are discussed. Previously unpublished data of an experiment by G.E. Schreiner, on the effects of massive iv 1% sodium chloride infusions in dogs poisoned orally with ethanol, are tabulated.

719. Koppanyi, T., and Maengwyn-Davies, G. D.
DRUGS USED IN ALCOHOL RESEARCH AND TREATMENT.
 In: Rabinowitz, Joseph L., and Myerson, Ralph M., eds. *Topics in Medicinal Chemistry, II*. New York: Interscience Publications pp. 155-183 (183 ref.), 1968.
 E – review – cross-tol. – DC (antidotal) – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – drug-dep. humans – mammals – other org. – acute admin. – chronic admin. – in vivo – in vitro – cardiovasc. – CNS – G.I. tract – glands – liver, kidney – metab. proc. – amphetamines – anesthetics – barbiturates – cardiovasc. agents – elect., water-bal. agents – hormones, hormone antag. – miscellaneous – stimulants – tranquilizers – *CAAAL-0 B-0950.

The pharmacology of alcohol, the interaction of alcohol with other compounds—CNS depressants and antidepressants (barbiturates, non-barbiturate hypnotics, opiates and opioids, tranquilizers, and CNS stimulants), hepatotoxic agents, oral hypoglycemic agents, diuretics, biogenic amines and hormones, and miscellaneous drugs—and the treatment of alcohol poisoning (acute inebriation, alcoholic coma, chronic alcoholism, and treatment of abstinence syndrome), are reviewed. Alcohol and aliphatic depressants (except aldehydes) reinforce the action of each other, and induce cross-tolerance. Synergism between short-acting barbiturates and alcohol seems greater than with long-acting malonyl ureas. All non-barbiturate hypnotics are proven or presumed to synergize with alcohol. Morphine and other potent analgesics increase alcohol toxicity. Synergism has been reported between alcohol and various tranquilizers. Chronic administration to rodents of some CNS depressants, such as chloroform and carbon tetrachloride, has produced a liver cirrhosis-like syndrome and a preference for alcohol. Some sulfonylureas, such as tolbutamide, produce a disulfiram-like intolerance to alcohol; similar effects have been found with tolazine, chloral hydrate, nitrofan derivatives, and *Coprinus atramentarius*. Hydrocortisone and some synthetic analogues, as well as glucagon, have been shown to accelerate alcohol metabolism. Alcohol consumption can be decreased with amphetamine, alloxan, estrogen, and metronidazole, and increased with other drugs, e.g., meprobamate. Isoniazid decreases alcohol toxicity, while dithiols increase alcohol sleeping time and lower the LD₅₀. Quinine increases the hypnotic effects, and arsenite increases the duration and intensity, of alcohol narcosis.

720. Korablev, M. V.
VLIHANIE POLISUL'FIDOV GRUPPY TIURAMA NA TECHENIE ISKHOD ALKOGOL'NOI INTOKSIKATSII. [The effect of polysulfides of the thiram group on the course and outcome of alcohol intoxication].
 Farmakol. Toksik. (Moscow), 22(3): 259-261 (7 ref.), 1959.
 R – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (sensit.) – mammals – acute admin. – chronic admin. – in vivo – CNS – unclass. ther. agents – *CAAAL-9029-D2 A-1296.

Mice weighing between 16-28 g received disulfiram, tetramethylthiuramdisulfide, sodium dimethyldithiocarbamate, or sodium diethyldithiocarbamate, once a day in doses of 200 mg/kg for a period of 4 days. 3 hr after the last dose of thiram compound, the mice received a 1:1 sol of 96% alcohol in

continuously increasing doses. From the results, curves of individual sensitivity of mice were plotted for each substance, and compared with controls receiving alcohol alone. The potentiating effects of the substances were indicated in increased sensitivity of mice towards alcohol, and in the longer duration of intoxication. The strongest effects were observed with antabuse, the potentiating effects of which were found to depend upon the dose of alcohol administered. The author concludes that all of the substances increase and prolong the effect of alcohol in the following order of potency: disulfiram, sodium dimethyldithiocarbamate, tetramethylthiuramdisulfide, and sodium diethyldithiocarbamate.

721. Kordecki, R., Reutt, H., and Rożkowski, K.
 EFPEKTY HEMODYNAMICZNE WSTRZĄSORODNYCH DAWEK HISTAMINY I
 BŁĘKITU TRYPANU W EPOJENIU ALHOKOLEM ETYLOWYM U PSÓW. [Hemodynamic
 effects of shock doses of histamine and trypan blue during ethyl alcohol intoxication in dogs].
 Rocz. Akad. Med. Marchewski (Białystok), 10: 79-89 (15 ref.), 1964.
 Po – ES – RS – exp. comp. – DC (decrease) – DC (add., infra-add., unsec. incr.) – mammals – acute
 admin. – in vivo – dose resp. – blood lev. – cardiovasc. – CNS – cardiovasc. agents –
 *CAAAL-11628-D2 A-0845.

Ethanol was administered iv in a 30% sol in doses designed to reach blood alcohol concentrations of 50, 100, 200, or 300 mg%, to 48 dogs anesthetized with chloralose. Shock, induced with histamine or trypan blue, was intensified when the 2 smaller doses of ethanol were given, i.e., arterial pressure fell lower, and took longer to return to normal. The 2 higher doses of ethanol, however, seemed to diminish the hemodynamic disturbances. The possibility that the stimulating effect of the smaller doses and the depressing effect of the larger doses of ethanol were responsible for these results is proposed.

722. Kósa, F.
 DATA ON THE DEGREE OF ALCOHOL INTOXICATION IN RATS EXAMINED WITH
 THE TILTING-PLANE METHOD.
 Quart. J. Stud. Alcohol (New Haven), 25: 253-261 (10 ref.), 1964.
 E – exp. comp. – DC (decrease) – DC (add., infra-add., unsec. incr.) – mammals – acute admin.
 – in vivo – blood lev. – mot. perform. – CNS – skel., muscle, skin – stimulants – *CAAAL-10310-J2
 A-0846.

Groups of 5 male rats were placed on a movable plane which was tilted through 90° in 5 sec, before and after the injection of 2 or 5 g of alcohol/kg in a 20% sol. The angles at which the rats slid down were recorded hourly, and compared with the blood alcohol values. The results showed that the sliding angle values were related to the regression line of the hourly-measured blood alcohol concentrations. When 0.04 and 0.05 g of pentamethazol/kg or 0.1 g of sodium phenylethylbarbiturate were given separately and together with alcohol, measurable differences in the sliding angle, showing antagonism and synergism of alcohol, respectively, were induced.

723. Kotoku, S.
 ZOKUZOKU NARU JÔKEN NO KETSUEKICHÛ ARUKÔRU NÔDO NI OYOBOSU
 EIKYÔ NI TSUITE. [The influence of various conditions on the blood alcohol concentration].
 Yonago Igaku Zasshi (Yonago Medical Association, Journal) (Yonago), 4: 206-215 (17 ref.),
 1953.
 J – ES – exp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp.
 – CNS – metab. proc. – nutritive agents – *CAAAL-0 A-1338.

In accordance with the theory, enunciated in 1945 by Shigeo Ogata, that the genesis of alcohol habituation lies in the changes in alcohol distribution in the body, and in the increase of the oxidizing

ability of alcohol, the effects of various conditions on the blood alcohol level (BAL) decline were investigated. The effects of levulose, vitamin B₁, potassium cyanide, caffeine, citrated ferrous oxide, vitamin C, cyclopal sodium, and malnutrition on the BAL were studied in rabbits administered various doses of alcohol. It was found that 3 cc/kg 5% levulose sc accelerated recovery from alcohol intoxication, as did 10 mg vitamin B₁ sc. 1 mg/kg potassium cyanide sc and 2% caffeine sc produced a more rapid than normal decline of the BAL. 10 mg/kg citrated ferrous oxide sc or 100 mg/kg vitamin C administration resulted in a slightly accelerated decline. Injection of 0.5 g cyclopal sodium into the hip muscle, however, failed to affect the BAL. When the weight of the animals was reduced by hunger to 2/3-3/5 of the normal weight, the BAL decline was slowed down.

724. Kraft, H. -G.

VERKEHRSTÜCHTIGKEIT NACH GEBRAUCH ZENTRALER STIMULANTIEN.

[Performance in traffic under the influence of central stimulating drugs].

Arzneimittelforschung (Aulendorf), 12(11): 1071-1074 (25 ref.),

1962.

G – ES – exp. – general – DC (decrease) – DC (unchanged) – med.-leg. – mot. vehic. – humans – blood lev. – mot. perform. – cardiovasc. – CNS – metab. proc. – respir. – amphetamines – stimulants – *CAAAL-0

A-0847.

The problem of fatigue is of special interest in traffic medicine. Fatigue can be treated by influencing the metabolism (vitamin C, lactose), by stimulating the circulation (coffee, amphetamines), or by directly affecting the CNS (caffeine, nicotine, amphetamines, ephedrine, strychnine). Fatigue caused by alcohol is difficult to compensate. Experiments on a driving simulator have shown that the performance after 0.5 mg/kg alcohol was not normalized by 0.2 g caffeine; it was first normalized by 9 mg methamphetamine, but afterwards the performance was again impaired, relative to the blood alcohol level.

725. Krantz, J. C., Jr.

THE PROBLEM OF MODERN DRUG INCOMPATIBILITIES.

Amer. J. Pharm. (Philadelphia), 139: 115-121 (1 ref.),

1967.

E – SEC – general – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – cardiovasc. – CNS – G.I. tract – barbiturates – tranquilizers – *CAAAL-0

B-0349.

The author discusses, among other incompatibilities, the incompatibility between alcohol and drugs. The alcohol-antabuse syndrome is well known, causing a fall in blood pressure, gastrointestinal distress, and faintness. The oral antidiabetic drugs, such as tolbutamide and chlorpropamide, and also nitroglycerin, elicit the same kind of syndrome. The synergism of the narcotic action of alcohol with some central nervous depressants may produce coma or death. In this group are the barbiturates, meprobamate, and chlorpromazine and similar phenothiazine derivatives.

726. Kratzsch, E.

ÜBER DIE WIRKUNG VON CYSTEIN, RUTABION UND BAL AUF DIE

CYANAMID-ALKOHOLVERGIFTUNG. [On the action of cysteine, rutabion and BAL on cyanamide-alcohol poisoning].

Dissertation, Medical Faculty of the University of Leipzig, East Germany, 33 pp. (45 ref.),

1956.

G – exp. cont. – case hist. – DC (antidotal) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – DC (sensit.) – humans – mammals – acute admin. – in vivo – acid-base, blood pH, elect. – *CAAAL-0

A-0848.

The author reviews the literature on interaction of calcium cyanamide (a fertilizer) and alcohol. A case of a young worker in a fertilizer factory, who was not sufficiently protected from inhaling calcium cyanamide dust, is described. 45 min after 2 sips of cognac, cyanosis of the face and hyperhidrosis

of the hands was observed. 1 cc cysteine hydrochloride was applied im, and, 30 min later, the subject had returned to normal. In an experiment with rabbits, the animals received 300 mg/kg cyanamide in 5% sol iv, alone or in combination with 1 g/kg alcohol in 20% sol iv. The animal receiving only cyanamide returned to normal after 48 hr; of the 3 animals receiving cyanamide and alcohol, 1 recovered after 72 hr, 1 died after 19 hr, and 1 died after 72 hr. In other animal experiments with mice and dogs, alcohol applied simultaneously increased the effect of cyanamide 2 to 5 times. Different animal species received cysteine hydrochloride, BAL (dimercaprol), or rutabion (rutin), but no effect on the poisoning was observed.

727. Krug, W.

ALKOHOL UND SCHLANGENGIFT. [Alcohol and snake venom].

Dissertation, Medical Faculty of the University of Marburg, Germany, 27 pp. (10 ref.), 1935.
G – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – in vitro – absorp., distrib., stor. – G.I. tract – *CAAAL-0 A-0849.

In controlled experiments on mice and guinea pigs, applications of 5% alcohol po had no effect on survival times from lethal or sub-lethal (66%) doses of snake venom administered sc. Alcohol clearly diminished symptoms of pain; the animals calmed down following alcohol administration, and did not react when touched in the otherwise sensitive injection area. In isolated guinea pig stomachs, alcohol (up to 20%) had no effect on the diffusion of saponin and digitonin. The author concludes that, aside from the symptomatic pain-relieving action, alcohol cannot be regarded as an antagonist to snake bite. Since serums are now available for treatment of snake bites, they should be used if possible.

728. Krull, G.

CORAMIN BEI ALKOHOLVERGIFTUNGEN. [Coramine in alcohol poisoning].

Munchen. Med. Wschr. (Munich), 84(50): 1987-1988 (9 ref.), 1937.
G – general – case hist. – DC (antidotal) – humans – cardiovasc. – CNS – respir. – stimulants – *CAAAL-0 A-0850.

A case is presented of a man who was found unconscious and admitted to hospital. The diagnosis was alcohol intoxication, serious circulatory failure, respiratory failure, and deep coma. The face was blue-red and bloated, respiration shallow, and the pulse hardly noticeable. He received initially 5 cc coramine (nikethamide) iv and 5 cc im. The patient reacted immediately—respiration became deeper, and the pulse fuller and more regular. After 10 min, he received another 5 cc iv (and started to sneeze more than 20 times), and, 15 min later, another 5 cc iv. 5 min later, he was able to get up and walk. After this experience, coramine was used in many similar cases, with the same good results. Heavy sneezing was always observed.

729. Kubička, J.

POZNÁMKY K OTRAVĚ HNÍKEM INKOUSTOVÝM (COPRINUS ATRAMENTARIUS BULL. EX FR.). [Notes on poisoning by the common inky cap (*Coprinus atramentarius* Bull. ex Fr.)].

Ceska Mykologie (Prague), 4: 62-65 (4 ref.), 1950.
C – general – DC (sensit.) – humans – cardiovasc. – *CAAAL-0 A-1297.

Coprinus atramentarius (inky cap) is an edible mushroom which becomes poisonous only when consumed in conjunction with alcohol. The poisoning appears within 20 min to 2 hr in the form of purple spots on the skin, increased body temperature, and quickening of the pulse (130-150/min). The author compares these characteristics with the effect of tetraethylthiuramdisulfide (antabuse) plus alcohol on the human organism. The similarities lead to the theory that the inky cap contains either antabuse or some other substance that forms an excess of acetaldehyde from the consumed alcohol.

The induced acetaldehyde poisoning can result in intolerance to alcohol, and thus the substance can be effective as a sobering-up agent. The author suggests commercial growing of the inky cap as a cheap source of antabuse, or for direct clinical use, since its effects are much faster, although not as pronounced, as those of antabuse.

730. Kudrin, A. N.

LECHENIE OSTROGO ALKOGOL'NOGO OTRAVLENIIA KOMBINATSIIAMI ANALEPTIKOV TSENTRAL'NOI NERVNOI SISTEMY. [Treatment of acute alcohol poisoning with combinations of analeptics of the central nervous system].

Farmakol. Toksik. (Moscow), 19(Suppl.): 56-57 (0 ref.),

1957.

R – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – CNS – stimulants – *CAAAL-8075-D2 A-0851.

2 sets of experiments were conducted on dogs. In the first series, 36 dogs were put into a deep coma by administration of 15 ml/kg 40% alcohol by stomach tube; they began to stand up 28-34 hr later, and some died if left untreated. When 5 different combinations of caffeine, corazole, picrotoxin, strychnine, cytisine, and distilled water were given sc 1, 2, and 3 hr after alcohol, the dogs began to stand up 8-12 hr after the alcohol. Most effective were mixtures of 0.1, 0.1, and 0.2 ml/kg, containing (in their respective order): 0.1, 0.063, and 0.5 g/ml caffeine; 0.1, 0.063 and 0.05 g/ml corazole; 0.0005, 0.00025, and 0.00025 g/ml picrotoxin; 0.005, 0.00025, and 0.00025 g/ml strychnine; 0, 0.0005, and 0.0005 g/ml cytisine; and 1.0, 1.0, and 1.0 g /ml distilled water. In the second series, dogs received 10 ml/kg 40% alcohol by stomach tube; they began to stand up 7-8 hr after the alcohol. When the combinations of analeptics were given sc, the animals began to walk 5-35 min after the injections. The addition of cytisine made little difference to the effectiveness of the mixture.

731. Kuenssberg, E. V.

SIDE-EFFECTS OF ETHCHLORVYNOL.

Brit. Med. J. (London), 2: 1610 (0 ref.),

1962.

E – general – DC (add., infra-add., unspec. incr.) – drug-dep. humans – cardiovasc. – sed., hypnot. – *CAAAL-10272-E8 A-0852.

The writer draws attention to the alarming consequences which can ensue if alcohol is consumed during treatment with ethchlorvynol. 2 cases are described in which the patient was found unconscious. In the first case, a 54 yr-old woman consumed 500 mg ethchlorvynol (unconsciousness lasted 40 min), and, in the second, a man of similar age consumed not more than 750 mg (the duration of unconsciousness required hospitalization). Both patients were former alcoholics with probable liver damage. They each denied having consumed alcohol, but the author considers that this was probably done, without his knowledge.

732. Kulisiewicz, T. A.

EFFECTS OF HYDROXYZINE (ATARAX, U.C.B. 4492) IN CASES OF PSYCHOMOTOR EXCITATION DUE TO ETHYL ALCOHOL INTOXICATION.

Conference of Antialcoholic Dispensary Workers, Warsaw, Poland, 5 pp. (8 ref.),

1959.

E – exp. – general – presentation – DC (antidotal) – drug-dep. humans – acute admin. – in vivo – mot. perform. – cardiovasc. – CNS – respir. – tranquilizers – *CAAAL-0 A-0853.

96 males and 6 females with acute ethanol intoxication, attended by a marked verbal and motor excitation, were treated at the Warsaw Detoxication Centre with im injections of hydroxyzine (atarax). The degree of intoxication was evaluated as slight (4 cases), medium (90 cases), or severe (8 cases). The dosage was 100 or 200 mg (up to 300 mg in very severe cases) hydroxyzine im. All cases showed complete disappearance of verbal and motor excitation within 10 to 90 min following drug intake. It is concluded that hydroxyzine should be the drug of choice in detoxication centres for

controlling the psychomotor excitation resulting from acute ethanol intoxication. Also abstracted in Kulisiewicz, Tadeusz A., *26th International Congress on Alcohol and Alcoholism, Abstracts*, pp. 324-325, Stockholm, Sweden, 1960.

733. Kulisiewicz, T. A.
EFFECTS OF HYDROXYZINE (ATARAX, U.C.B. 4492) IN CASES OF PSYCHOMOTOR EXCITATION DUE TO ETHANOL INTOXICATION.
In: *Twenty-sixth International Congress on Alcohol and Alcoholism, Abstracts* Stockholm, Sweden, August 1-5, pp. 324-325 (0 ref.), 1960.
E – abst. – general – presentation – DC (antidotal) – drug-dep. humans – acute admin. – in vivo – mot. perform. – cardiovasc. – CNS – respir. – tranquilizers – *CAAAL-0 A-0854.

96 males and 6 females with acute ethanol intoxication, attended by a marked verbal and motor excitation, were treated at the Warsaw Detoxication Centre with im injections of hydroxyzine (atarax). The degree of intoxication was evaluated as slight (4 cases), medium (90 cases), and severe (8 cases). The dosage was 100 or 200 mg (up to 300 mg in very severe cases) hydroxyzine im. All cases showed complete disappearance of verbal and motor excitation within 10 to 90 min following drug intake. It is concluded that hydroxyzine should be the drug of choice in detoxication centres for controlling the psychomotor excitation resulting from acute ethanol intoxication. More detailed information is in Kulisiewicz, T. A., *Conference of Antialcoholic Dispensary Workers*, Warsaw, Poland, 5 pp. 1959.

734. Kulisiewicz, T. A.
KILKA UWAG O TZW. „ŚRODKACH TRZEŹWIACYCH“. [Some remarks on so-called “sobering agents”].
Walka z Alkoholizmem (Warsaw), 9(3): 8-9 (1 ref.), 1961.
Po – general – DC (decrease) – DC (unchanged) – humans – blood lev. – absorp., distrib., stor. – metab. proc. – amphetamines – elect., water-bal. agents – hormones, hormone antag. – stimulants – *CAAAL-0 A-0855.

The various reputed sobering agents are discussed in the light of research conducted in 1955 at the Institute for Medical Research, University of Bonn—see: Elbel, Herbert, *Zentralblatt für Verkehrsmedizin, Verkehrs-Psychologie und Angrenzende Gebiete*, 1: 89-92, 1955; and Paulus, Walter, and Mallach, H. J., same journal, 1: 92-95, 1955. Certain factors involved in alcohol antagonism, absorption, elimination, and oxidation are mentioned, and brief reference is made to the following substances: insulin, levulose, dinitrophenol, caffeine, pervitin, and “contra” or “stop”.

735. Kup, G.
LUGMÉRGEZÉS HATÁSA ALKOHOLLAL ÁTÍTATOTT EMÉSZTŐTRACTUSBAN.
[Effect of caustic soda poisoning on digestive tract saturated with alcohol].
Gyógyászat (Budapest), 76: 88 (0 ref.), 1936.
H – SEC – general – DC (unspec.) – med.-leg. – humans – G.I. tract – *CAAAL-0 A-1298.

A case of murder or possible suicide was investigated. The subject, a 68 yr-old man, was found dead from caustic soda poisoning. The post-mortem showed clearly the effect of the lye in the digestive tract. The stomach was corroded up to the pylorus, and only spots of mucous membrane were left unharmed. However, other parts of the gastrointestinal tract remained well preserved, even 3 1/2 weeks after death. The subject had consumed a considerable amount of alcohol before taking the caustic soda, and the alcohol preserved the gullet and the stomach. Only this fact made it possible to determine that suicide, and not murder, had occurred.

736. Kutob, S. D., and Plaa, G. L.

THE EFFECT OF ACUTE ETHANOL INTOXICATION ON CHLOROFORM-INDUCED LIVER DAMAGE.

J. Pharmacol. Exp. Ther. (Baltimore), 135: 245-251 (25 ref.), 1962.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – CNS – liver, kidney – anesthetics – *CAAAL-10697-B2 A-0856.

Pentobarbital (45 mg/kg ip) sleeping time, bromsulphthalein (BSP) retention, and liver succinic dehydrogenase activity (SDA) were measured in rats which were pretreated with single or repeated doses of ethanol, and then given a minimally hepatotoxic dose of chloroform. Each test involved 4 groups of animals which received no treatment, 2.5 or 5 g/kg ethanol po, 0.08 ml/kg chloroform sc, or ethanol plus chloroform. It was found that mice receiving chloroform 12, 15, or 24 hr after 5 g/kg ethanol showed an enhanced response to chloroform; 12 hr after 2.5 g/kg ethanol, however, the response to chloroform was unaltered. Animals given chloroform 15 and 24 hr after a single administration of 5 g/kg ethanol showed a significant increase in the incidence of elevated BSP levels; this was also true for mice given ethanol once/day 2 or 4 days prior to chloroform. When chloroform was given 12 or 24 hr after 5 g/kg ethanol, SDA was significantly depressed. When ethanol pretreatment preceded chloroform by 48 hr, and when 2.5 g/kg ethanol was given 12 hr prior to chloroform, SDA was not depressed. Histological findings were consistent with the results. Chemical analysis demonstrated that the increased incidence of abnormal liver function is probably due to an ethanol-induced increase in liver lipids, resulting in a higher chloroform retention.

737. Laborit, H., Jouany, J. M., Gerard, J., and Drouet, J.

SUR UN SYNDROME EXPERIMENTAL D' "EXCITATION-HYPOTONIE". [An experimental "excitation-hypotonia" syndrome].

Arch. Int. Pharmacodyn. (Gand), 131(1-2): 151-163 (7 ref.), 1961.
 F – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – CNS – metab. proc. – nerv. syst. – skel., muscle, skin – anesthetics – anti-infectants – miscellaneous – *CAAAL-9481-B2 A-0857.

An "excitation-hypotonia" syndrome, characterized by tremors, muscular jerks, and decrease in tonus, was induced in rats by substances with an unsubstituted benzene ring, containing an OH or NH₂ group. The compounds having such an effect were: hydroquinol, pyrocatechol, resorcinol, o-phenylenediamine, p-phenylenediamine, o-aminophenol, m-aminophenol, quinhydrone, phenol, aniline, α -naphthol, and β -naphthol; all were administered ip. It was found that ip injections of 4 ml/kg 90% ethanol prevented the syndrome in all cases, as did anesthetic doses of sulfuric ether.

738. Lachnit, V., and Pietschmann, H.

ACTIVITY OF SERUM GLUTAMIC-OXALOACETIC-TRANSAMINASE AND ALDOLASE IN WORKERS EXPOSED TO HALOGENATED HYDROCARBONS.

Industr. Med. Surg. (Chicago), 29: 523-525 (17 ref.), 1960.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – chronic admin. – in vivo – blood comp., sites, lymph – liver, kidney – metab. proc. – *CAAAL-0 A-0858.

In workers acutely or chronically intoxicated after exposure to halogenated hydrocarbons, measurements were made of serum glutamic-oxaloacetic transaminase (SGOT) and aldolase activities, as indices of liver function. The effect of alcohol upon enzyme activity after exposure to trichloroethylene was also investigated. 2 groups of human subjects, 1 group exposed to trichloroethylene and 1 not exposed, were given 100 ml 40% ethanol, and the enzyme activity was tested 36 hr later. It was found that the group not exposed to trichloroethylene had normal enzyme activity, whereas, in the exposed group, 6 persons showed a marked increase of SGOT, 2 showed a moderate increase, and 9 exhibited

uncharacteristic oscillations toward the upper or lower levels; there were no oscillations of aldolase activity beyond the error limit, with the exceptions of 1 out of 6 subjects.

739. Laiho, K., Isokoski, M., and Alha, A.
 TOD IN DER SAUNA NACH EINNAHME VON CHININ UND ALKOHOL. [Death in the sauna following ingestion of quinine and alcohol].
 Arch. Toxik. (Berlin), 21: 352-354 (9 ref.), 1966.
 G – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – anti-infectants – *CAAAL-0 B-0350.

A 17 yr-old girl, who wanted to induce an abortion, went to an overheated sauna, drank 0.5 l strong wine, and took 1 tablet quinine sulfate (0.3 g). After 45 min, the girl became unconscious and, 30 min later, was pronounced dead after admission to hospital. The blood alcohol level was 1.06°/oo, and the quinine content 0.66 mg/100 ml blood. It is concluded that the girl must have taken more than 0.3 g quinine, since the lethal quinine dose in humans is about 2.4 mg/100 ml, and the lethal alcohol dose about 4.0-5.0°/oo. It is believed that a synergism between quinine and alcohol, intensified by hyperthermia, caused the death.

740. Lamson, P. D., Gardner, G. H., Gustafson, R. K., Maire, E. D., McLean, A. J., and Wells, H. S.
 THE PHARMACOLOGY AND TOXICOLOGY OF CARBON TETRACHLORIDE.
 J. Pharmacol. Exp. Ther. (Baltimore), 22(4): 215-288 (20 ref.), 1924.
 E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – absorp., distrib., stor. – blood comp., sites., lymph – G.I. tract – liver, kidney – anti-infectants – *CAAAL-0 A-0859.

A careful and comprehensive study on carbon tetrachloride (CCl₄) is presented. In 1 experiment, 25 dogs were used to test the effect of ethanol on CCl₄ toxicity. 5 controls received 4 ml/kg of 97% alcohol, 8 dogs received 1 ml/kg alcohol plus 4 ml/kg CCl₄ po, 8 were given 4 ml/kg ethanol and 4 ml CCl₄ po, and 4 received 4 mg/kg alcohol plus 10 ml CCl₄ po. In all cases in which alcohol and CCl₄ were given together, the degree of toxicity of the CCl₄ was increased considerably, and mortality was much higher. From the many blood samples necessary for the phenoltetrachlorophthalein test, it was found that blood from CCl₄-intoxicated animals clotted much more slowly than normal; this was especially marked after the alcohol-CCl₄ combinations.

741. Lamson, P. D., and Wing, R.
 EARLY CIRRHOSIS OF THE LIVER PRODUCED IN DOGS BY CARBON TETRACHLORIDE.
 J. Pharmacol. Exp. Ther. (Baltimore), 29(1): 191-202 (4 ref.), 1926.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. – in vivo – dose resp. – liver, kidney – anti-infectants – *CAAAL-2246-G2 A-0860.

The following doses were administered to dogs: 2 dogs—3 cc carbon tetrachloride (CCl₄), 36 times in 15 weeks; 2 dogs—25 cc CCl₄, 17 times in 8 weeks; 1 dog—25 cc CCl₄, 37 times in 15 weeks; 1 dog—25 cc CCl₄, 44 times in 17 weeks; 2 dogs—25 cc 50% ethanol plus 3 cc CCl₄, 38 times in 17 weeks; 1 dog—50 cc 50% ethanol, 15 times in 7 weeks; and 1 dog—25 cc 50% ethanol, 16 times in 7 weeks, then 50 cc 50% ethanol, 13 times in 6 weeks. The CCl₄ was administered by stomach tube. It was found that, despite continued administration of the drugs, the animals were apparently in perfect condition, and most actually gained wt. At the end of the period of observation, the dogs were killed and autopsied. The results showed that only the animals receiving ethanol alone had no liver lesions; it was found that, in dogs, the toxicity of a small dose of CCl₄ is not necessarily increased by the addition of ethanol—this was unexpected in view of the great increase in toxicity after large amounts of CCl₄ taken with ethanol. There was very little difference in the livers of animals receiving 3 cc or 25 cc CCl₄ only, and in those receiving 3 cc CCl₄ plus 25 cc 50% ethanol.

742. Lamson, P. D., and Wing, R.
THE EFFECT OF CARBON TETRACHLORIDE AND ALCOHOL ON THE ACID-BASE BALANCE OF THE BLOOD.
 J. Biol. Chem. (Baltimore), 69: 349-355 (13 ref.), 1926.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – acid-base, blood pH, elect. – blood comp., sites, lymph – liver, kidney – anti-infectants – *CAAAL-0 A-0861.

Tests were made on dogs to determine whether the administration of carbon tetrachloride (CCl_4), alone or in combination with alcohol, might produce an acidosis which can be counter-balanced by alkali, and to compare the effect on the acid-base balance of CCl_4 inhalation with that of other volatile anesthetics. In 1 test, 5 dogs received 4 cc/kg CCl_4 and alcohol in combination po. The CCl_4 -alcohol combination produced no change in the acid-base balance, except in the terminal stage of intoxication, and a considerable period of time after the appearance of symptoms.

743. Lamson, P. D., Minot, A. S., and Robbins, B. H.
THE PREVENTION AND TREATMENT OF CARBON TETRACHLORIDE INTOXICATION.
 J.A.M.A. (Chicago), 90(5): 345-349 (11 ref.), 1928.
 E – general – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – blood comp., sites, lymph – G.I. tract – liver, kidney – anti-infectants – *CAAAL-0 A-0862.

In a general discussion illustrated with case histories, the author discusses carbon tetrachloride (CCl_4) intoxication, with reference to the effect of alcohol on toxicity, ascariids as a complicating factor, the effect of food, the effect of an insufficient calcium supply, and prevention and treatment in general. The signs and symptoms of CCl_4 intoxication in alcoholics or after alcohol consumption are extremely severe—vomiting is violent and continuous, and there are almost always hemorrhages into the gastro-intestinal tract; these hemorrhages cause the patient to vomit either bright or changed blood, and to pass bloody stools. Jaundice may be very severe after 48 yr of age, and the urine is scanty and blood-stained. 3 case histories (2 fatal), in which alcohol was a factor, are reported.

744. Landabure, P. B., Delbue, C., Alvariñas, C., Serantes, N., Davalos, R. C., Waisman, G., Cabarro, A., Dussel, E., Cicchitti, F., and Lored, A.
SULFONYLUREA DRUGS IN THE TREATMENT OF DIABETES MELLITUS.
 Ann. N.Y. Acad. Sci. (New York), 74: 794-809 (13 ref.), 1958-59.
 E – SEC – general – DC (sensit.) – humans – dose resp. – cardiovasc. – skel., muscle, skin – hormones, hormone antag. – *CAAAL-0 A-1299.

In this extensive clinical investigation of the use of chlorpropamide in the treatment of diabetes mellitus, alcohol is implicated in 1 of the reported side-effects. The data presented was gathered from 130 patients, 30 (23%) of whom were studied while they were hospitalized, and 100 of whom (77%) were observed in the outpatient department. The drug was administered in most cases in 2 doses (1 after breakfast and 1 after lunch), and the dosage usually ranged from 0.1-2.0 g/day. In general, side effects were related to dosage, appearing when the dosage was 1 g/day or higher, but only rarely with a dosage of 0.5 g/day. In 14 (10%) cases, flushing was reported as a side effect of chlorpropamide administration. The ingestion of alcohol was implicated in 2 of these cases, since the intense flushing disappeared when alcohol consumption was reduced. The authors interpret the flushing to be the result of a vasodilating effect, and not an allergic reaction.

745. Landauer, A. A., Milner, G., and Patman, J.
ALCOHOL AND AMITRIPTYLINE EFFECTS ON SKILLS RELATED TO DRIVING BEHAVIOR.

Science (Washington), 163(3874): 1467-1468 (6 ref.), 1969.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – acute admin. – in vivo
 – blood lev. – other drug lev. – CNS – antidepressants – *CAAAL-0 B-0351.

21 healthy, young adults were tested after having been given combinations of amitriptyline (0.8 mg/kg), placebo, and alcohol (in amounts sufficient to produce a blood alcohol level of 0.08%). 3 motor-skill tests were performed before and after drug administration. The results indicate that amitriptyline, a tricyclic antidepressant commonly prescribed for out-patients, potentiates the effect of alcohol. The interaction was significant after only 1 dose of amitriptyline, and was especially pronounced after 2 doses (interval between doses—12 to 15 hr).

746. Láng, S., and Schlick, B. von
 ÜBER DIE BEEINFLUSSBARKEIT DES ALKOHOLUMSATZES IM ORGANISMUS. [The possibility of influencing alcohol metabolism in the organism].
 Zeitschrift für die Gesamte Experimentelle Medizin (Berlin), 99: 81-84 (7 ref.), 1936.
 G – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo
 – blood lev. – absorp., distrib., stor. – metab. proc. – hormones, hormone antag. – stimulants –
 *CAAAL-1173-A2 A-0863.

Dogs received the following after 24 hr fasting: 2 dogs—0.5 cc 96% alcohol/kg in 9.6% sol by stomach tube, 1 dog—intracardial injection of 0.5 cc 96% alcohol/kg, and 1 dog—0.3 cc saline. Alcohol levels were determined by the Widmark micromethod. Insulin 20 IU, 2 mg thyroxin, or 0.2 g caffeine sodium benzoate in 20% sol were then given immediately after alcohol (except when alcohol was given iv, in which case insulin was given 2 hr previously). 1 of the dogs received 2 mg thyroxin every second day for 1 week prior to the experiment. It was found that insulin and thyroxin did not increase the β values, nor did it change the blood alcohol curve. After caffeine, the concentration of alcohol in the blood was greater in animals which received po alcohol than in the one which received iv alcohol. This may indicate that caffeine accelerates alcohol absorption. The later course of the blood alcohol curve was parallel to the control.

747. Lange, H. -J.
 NOLUDAR, EIN NEUES SCHLAFMITTEL. [Noludar, a new hypnotic].
 Med. Klin. (Munich), 51(8): 303-304 (3 ref.), 1956.
 G – SEC – exp. – DC (add., infra-add., unspec. incr.) – CNS – sed., hypnot. – *CAAAL-0
 A-0864.

Noludar (methypylone) was tested in 118 volunteers and 300 patients. It was found to be an effective sleep-inducing sedative with no side effects. As a day-time sedative, it also had an euphoric effect, and can be used with success in depressed patients. A dose of 0.2 to 0.4 g has a good hypnotic effect. A synergistic action with alcohol was observed. Low amounts of beer and noludar produced pronounced euphoria.

748. Langman, M. J. S.
 FAECAL BLOOD-LOSS AFTER SODIUM ACETYLSALICYLATE TAKEN WITH
 ALCOHOL.
 Lancet (London), 2: 54-55 (3 ref.), 1969.
 E – exp. – DC (unspec.) – humans – acute admin. – in vivo – cardiovasc. – G.I. tract – analg.,
 antipyret. – *CAAAL-0 B-0533.

The author contributes to the discussion surrounding the paper by Ian A.D. Bouchier and H.S. Williams (Lancet, 1 (7587): 178-180, 1969) on faecal blood-loss after combined ingestion of sodium acetylsalicylate and alcohol. He states that the essential problem is whether a blood-loss which

increases by 0.5 ml daily when an alcohol and buffered-aspirin period is compared with an alcohol plus placebo period, can be viewed as the same as that which might be expected from ordinary aspirin, with or without alcohol. In a recent paper, the amount of blood-loss following the use of unbuffered aspirin preparations was found to be never less than 1.0 ml/day. Another paper by Kerry Goulston and Allan R. Cooke (Brit. Med. J., 4(5632): 664-665, 1968) has reported that the mean blood-loss of 3.2 ml/day produced by aspirin alone rose to 5.3 ml/day when alcohol was added. It is concluded that buffered aspirin preparations probably cause less occult micro-bleeding than those which are unbuffered. Insufficient evidence is available to implicate combined alcohol and aspirin ingestion in acute gastrointestinal hemorrhage.

749. Larsen, J. A., and Madsen, J.

INHIBITION OF ETHANOL METABOLISM BY ORAL ANTIDIABETICS.

Proc. Soc. Exp. Biol. Med. (New York), 109: 120-122 (17 ref.), 1962.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – hormones, hormone antag. – *CAAAL-10005-A2 A-1300.

The effect of oral antidiabetics on ethanol metabolism was studied in cats. A priming iv dose of ethanol produced a plasma concentration of between 50-150 mg/l, which was maintained by continuous iv infusion. After diffusion equilibrium of ethanol had been established (1 hr), arterial blood samples were taken every 30 min for 4 hr, and determinations of plasma concentration of ethanol and plasma glucose were made. After 2 hr of sampling, the compound under study was slowly injected iv in an amount of 100 mg/kg. The disappearance rate of ethanol was determined before and after injection. In all 8 experiments with tolbutamide, the ethanol disappearance rate was reduced, on the average, by 34%, at a time when the effect on blood sugar was insignificant. The tolbutamide metabolite, N-(4-hydroxymethyl-benzolsulphonyl)-N'-n-butylurea, also inhibited ethanol metabolism, without having any hypoglycemic effect. Only 1 of 3 cats treated with carbutamide showed any inhibition of ethanol. It is concluded that tolbutamide (100 mg/kg) injected iv inhibits ethanol metabolism in cats.

750. Larsen, J. A.

THE EFFECT OF OXYGEN BREATHING AT ATMOSPHERIC PRESSURE ON THE METABOLISM OF GLYCEROL AND ETHANOL IN CATS.

Acta Physiol. Scand. (Stockholm), 73: 186-195 (25 ref.), 1968.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – acid-base, blood pH, elect. – cardiovasc. – liver, kidney – metab. proc. – nutritive agents – *CAAAL-13307 B-0534.

Oxygen toxicity in glycerol and ethanol metabolism was studied in chloralose-anesthetized cats (70 mg/kg chloralose). Following priming doses to bring the glycerol and ethanol concentrations to about 7 mM and 3 mM, respectively, a constant infusion of glycerol or glycerol plus ethanol was administered to the rats at the rate of 0.16 ml/min. After a 1 hr equilibration period, blood samples were taken and analyzed every 10-15 min. During simultaneous infusion of glycerol and ethanol, the mean elimination rate of glycerol decreased from 53 to 37 moles/kg/min when oxygen was breathed in place of air, whereas the ethanol elimination rate remained unchanged. The addition of glycine (0.5 g/kg iv) increased the elimination rate of both glycerol and ethanol, but oxygen breathing again reduced glycerol elimination, and also reduced ethanol elimination to a lesser extent. The glycerol elimination rate was not reduced by oxygen when glycerol was infused alone. It is concluded that the demonstrated effect of oxygen on glycerol metabolism is highly dependent on the simultaneous metabolism of ethanol, and may be explained by assuming that oxygen at high pressures inhibits the function of specific dehydrogenases responsible for the oxidation of ethanol and the production of adenosine triphosphate.

751. Läubli, E.
 ANTABUSÄHNLICHE WIRKUNG VON IRGAPYRIN. [Antabuse-like effect of irgapyrin].
 Schweiz. Med. Wschr. (Basel), 84(46): 1281-1284 (20 ref.), 1954.
 G – exp. comp. – DC (sensit.) – humans – acute admin. – in vivo – blood lev. – other drug lev. –
 mot. perform. – psychol. perform. – absorp., distrib., stor. – cardiovasc. – metab. proc. – unclass. ther.
 agents – *CAAAL-7221-A15 A-1415.

The combined effects of irgapyrin (aminophenazone + phenylbutazone) were investigated in 5 fasting men. The subjects on 3 separate occasions were given 1 of the following administrations: 3 bottles of beer (50-55 g absolute alcohol), 3 x 0.5 g irgapyrin during 8 hr, or the same alcohol and irgapyrin doses in combination. Irgapyrin caused a considerable delay in the rate of alcohol absorption, with lower and delayed maximal blood alcohol levels. However, contrary to expectations, the rate of elimination of alcohol was not affected. Breath analyses after the combined administration revealed a 2-10-fold increase of the acetaldehyde level. Alcohol alone caused euphoria, motoric manifestations, and, later, fatigue. Despite the lower blood alcohol levels after irgapyrin administration, the symptoms were more pronounced—headache, aversion to alcohol, nausea, fatigue, depression, apathy, paleness, pulse irregularities, sweating, and, in 3 cases, vomiting followed by collapse. It is not yet clear which of the components, aminophenazone or phenylbutazone, inhibit acetaldehyde oxidation; probably both have this effect. Similar effects have been reported with aminophenazone. Possibly other pyrazole derivatives produce alcohol intolerance; however, they may cause allergies.

752. Läubli, E.
 FRUCHTSÄFTE UND BLUTALKOHOLGEHALT. [Fruit juices and blood alcohol content].
 Praxis (Berlin), 43: 5-7 (5 ref.), 1954.
 G – exp. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – elect., water-bal. agents
 – *CAAAL-0 A-0865.

To test the effect of fruit juice on the blood alcohol level, 6 persons were each given 1 litre of non-alcoholic apple or grape juice/day for two days. The blood-alcohol content, as well as the alcohol content of the beverages, were quantitatively and qualitatively determined. It was established that the apple and grape juices contained between 0.05 and 0.5% alcohol. Careful determination did show a minute increase of the blood alcohol level, but this was so slight as to be well within the official 0.7% margin of error.

753. Läubli, E.
 ANTABUSÄHNLICHE WIRKUNG VON IRGAPYRIN. [Antabuse-like effect of irgapyrin].
 Schweiz. Med. Wschr. (Basel), 85: 94 (0 ref.), 1955.
 G – general – DC (sensit.) – humans – cardiovasc. – metab. proc. – *CAAAL-7221-A15 A-1416.

In an open letter, A. Meyer comments on Läubli's article (in Schweiz. Med. Wschr., 84: 1281-1283, 1954) on the disulfiram-like properties of irgapyrin (aminophenazone + phenylbutazone). Not only pyrazole derivatives have this effect. Egressin, which is not chemically related to irgapyrin or disulfiram, produces an intolerance reaction after either alcohol or nicotine consumption. Also, heavy smoking increases intolerance to alcohol. The fact that alcoholics are susceptible to pellagra indicates the possibility that there is a link with the PP factor, a coenzyme of diphosphopyridine nucleotide, which promotes acetaldehyde decomposition. The disulfiram treatment is a psychological one, creating Pavlovian conditioned reflexes. It accomplishes the same goal as prolonged institutionalization, without the enormous cost of the latter. Läubli replies that the effects of smoking on alcohol tolerance were in fact examined, but the individual differences which were found were so great that no general conclusions could be drawn. As for conditioned reflex, apomorphine can be used, but, in the case of disulfiram, the symptoms are due to actual toxic products of the disulfiram-alcohol interaction.

754. Lawton, M. P., and Cahn, B.

THE EFFECTS OF DIAZEPAM (VALIUM) AND ALCOHOL ON PSYCHOMOTOR PERFORMANCE.

J. Nerv. Ment. Dis. (Baltimore), 136(6): 550-554 (2 ref.), 1963.
 E – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – mot. perform.
 – psychol. perform. – CNS – tranquilizers – *CAAAL-10940-J1 A-0178.

20 human subjects each received, on different occasions, placebo, placebo plus alcohol (3 oz 100 proof vodka), placebo plus valium (5 mg 3 times/day for 2 days prior to test, plus 5 mg on day of test), or alcohol plus placebo, according to a replicated Latin square design, random administration. A battery of 4 psychological and psychomotor tests were given 1.25 hr after alcohol on days 4, 8, 12, and 16; blood alcohol was determined 1 and 4 hr after alcohol. The results showed a small but statistically significant tendency for psychomotor performance to be influenced by valium medication, either with alcohol or placebo drink. There was no evidence to suggest a potentiating decrement of performance with a combined dosage of valium and alcohol.

755. Leaf, G., and Zatman, L. J.

A STUDY OF THE CONDITIONS UNDER WHICH METHANOL MAY EXERT A TOXIC HAZARD IN INDUSTRY.

Brit. J. Industr. Med. (London), 9: 19-31 (56 ref.), 1952.
 E – exp. cont. – DC (decrease) – humans – mammals – acute admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – G.I. tract – metab. proc. – respir. – alcohols – *CAAAL-0 A-0866.

Experiments were conducted on human subjects to test the absorption and elimination of methanol. Several tests were made on the effect of ethanol on methanol absorption. Simultaneous ingestion of 4.0 ml methanol and 15 ml ethanol caused a marked elevation of peak methanol concentration. 15 ml ethanol, given 5 hr after 4.0 ml methanol, arrested the decline in body methanol concentration, which returned to its original rate after 2 hr. 15 ml ethanol, 4 1/2 hr after 4.0 ml methanol, followed by 4 doses of 7.5 ml ethanol at 1/2 hr intervals, arrested methanol elimination while adequate ethanol concentration remained in the body. After 15 ml ethanol taken simultaneously with 4.0 ml methanol, followed by hourly doses of 10 ml ethanol for 7 hr, body methanol concentration remained at a high level, and declined slowly; about 2 hr after cessation of ethanol administration, the decline in methanol concentration accelerated to control levels, indicating the release from inhibition of the chief mechanism of elimination. In all tests, ethanol reduced the rate of elimination of methanol by up to 90%, due to inhibition of methanol oxidation, and it is thus concluded that ethanol therapy is effective in methanol poisoning.

756. LeBlanc, E.

METHODOLOGICAL STUDIES ON THE MEASUREMENT OF ETHANOL INTOXICATION AND ACQUIRED TOLERANCE IN RATS.

M.Sc. Thesis, Department of Pharmacology of the University of Toronto, Ontario, Canada, 124 pp. (94 ref.), 1968.
 E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – dose resp. – barbiturates – *CAAAL-0 B-0352.

To develop a psycho-physiological model of alcohol-type tolerance suitable for use in a biochemical investigation of the basic mechanisms, practical techniques for the measurement of tissue level of ethanol, and for the measurement of drug effect in relation to dose and tissue level, were developed and validated. To further validate the technique, the scope of the investigation was broadened to include studies of other drugs, e.g., barbiturates, as well as drug interactions. In 1 experiment, dose-response curves were obtained for pentobarbital and alcohol which indicated that the former is 100 times more potent than the latter. 3 groups of 4 rats each then received 7, 8.5, or 10 mg/kg

pentobarbital, combined with 700, 850, or 1000 mg/kg alcohol, respectively. It was found that the dose-response curve for the alcohol-pentobarbital combination was almost exactly superimposable on the curves of the drugs administered individually, suggesting that the combined effect is merely additive, at least in the dose range tested.

757. Le Breton, R., Rondepierre, J. -J., Ropert, R., and Nizard, I.
 DE L'ALCOOLÉMIE CHEZ DES MALADES EN COURS DE TRAITEMENT
 CHIMIOTHÉRAPIQUE. [The blood alcohol level in patients under chemotherapeutic
 treatment].
 Ann. Medicopsychol. (Paris), 120/1: 755-759 (1 ref.), 1962.
 F – exp. comp. – DC (unchanged) – humans – chronic admin. – in vivo – blood lev. – CNS –
 antidepressants – barbiturates – sed., hypnot. – tranquilizers – *CAAAL-10238-U1 A-0867.

Blood alcohol determinations were carried out in 16 patients who were under treatment for periods of 14-62 days with a variety of neuroleptic drugs and sedatives. In 7 patients, the results were negative, and, in 9, none was above 0.1 g/l. The conclusions of Certhoux, J., and Ramet, M., (Ann. Medicopsychol. (Paris), 120/1: 359-364, 1962), that drugs, such as tranquilizers, make blood level values found according to the official method of determination erroneous, are thus refuted.

758. Le Breton, R., Le Bourhis, J., and Garat, J.
 UN CAS D'EMPOISONNEMENT CRIMINEL PAR LE TRICHLORÉTHYLÈNE. [A case of
 criminal poisoning by trichloroethylene].
 Ann. Med. Leg. (Paris), 43(3): 281-283 (0 ref.), 1963.
 F – general – DC (add., infra-add., unspec. incr.) – med.-leg. – post-mort. – drug-dep. humans – blood
 lev. – absorp., distrib., stor. – cardiovasc. – CNS – G.I. tract – liver, kidney – nerv. syst. – anesthetics
 – *CAAAL-0 A-1417.

A case of fatal poisoning by forced inhalation of trichloroethylene is described. The victim was a 35 yr-old man who was known to be an inveterate alcoholic, and who had several times been hospitalized for delirium tremens. On the morning after a night of drinking, he was found dead, and the examining physician, knowing the habits and poor state of health of the victim, determined that death was due to natural causes. An anonymous tip, however, led to an inquiry and autopsy. The autopsy, conducted 4 days after death, revealed acute pulmonary edema, and the presence of a brownish liquid in the stomach, bronchia, and trachea. The body was then examined by a toxicologist, who found quantities of trichloroethylene in the blood (30 mg/l), brain (45 mg/kg), liver (33 mg/kg), kidneys (25 mg/kg), and lungs (45 mg/kg), as well as a blood alcohol concentration of 2 g/l. The succeeding investigation revealed that, while the man was drunk, the wife of the victim had overpowered him, and had then held a cotton wad soaked with trichloroethylene against his mouth until no movement could be seen. Shortly afterwards, the man died. The author notes that the effect of the poison was definitely enhanced by the intoxicated state of the victim, but adds that the amount of the poison was alone sufficient to cause death.

759. Le Breton, R., and Garat, J.
 SUICIDES PAR LES DÉRIVÉS BARBITURIQUES ASSOCIÉS À L'ALCOOL ÉTHYLIQUE.
 [Suicides by barbiturate derivatives combined with ethyl alcohol].
 Ann. Med. Leg. (Paris), 45: 78-80 (0 ref.), 1965.
 F – stat. surv. – DC (add., infra-add., unspec. incr.) – humans – blood lev. – barbiturates – sed.,
 hypnot. – *CAAAL-0 B-0353.

Statistics over a 10-yr period of 253 cases of suicide with barbiturates were analyzed. 78 (30%) of the cases involved interaction with alcohol. The barbiturates found in combination with alcohol were: gardenal (42%), veronal (2%), soneryl (14%), immenonocet (22%), eunocet (8%), nembutal (8%),

seconal (2%), and phanodorm (2%). Of the cases of simple barbiturate poisonings, 41.2% were men and 58.8% women, whereas, of the barbiturate-alcohol suicides, 71.8% were men and 28.2% were women. With respect to the combined poisonings, the average blood alcohol levels were 1.35 g/l for men and 1.61 g/l for women, which fact is explained by the authors as being due to a more rapid death in women. In contrast to this, it was found that the average blood barbiturate levels were, for women—47 mg/l with alcohol and 46 mg/l without, whereas, for men, they were 54.8 mg/l with alcohol and 36.5 mg/l without. The greater variation in barbiturate levels for men is ascribed to a more rapid onset of death in the case of combined poisonings. The authors conclude that, in cases of combined poisonings, both the higher barbiturate levels in men and the higher alcohol levels in women indicate a greater resistance by men to the 2 substances taken together.

760. Lecoq, R., Chauchard, P., and Mazoué, H.
RECHERCHES SUR LE MÉCANISME D'ACTION DE QUELQUES SUBSTANCES PRÉCONISÉES DANS LE TRAITEMENT DE L'ALCOOLISME CHRONIQUE. [Studies on the mechanism of action of some substances recommended in the treatment of chronic alcoholism].
 Thérapie (Paris), 4: 182-195 (23 ref.), 1949.
 F – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – nerv. syst. – alcohols – amphetamines – anticonvulsants – cardiovasc. agents – gastrointest. agents – nutritive agents – unclass. ther. agents – *CAAAL-5415-D2 A-0868.

In rats, chronaxies were measured on nerves of the ear; results were expressed in chronaxic capacity. The different effects of ethanol when administered by various routes are described. Tables are presented showing the effects of alcohol by various routes, and the effects of the following, alone or in combination with alcohol: nicotinamide, vitamins B₃ and B₄, antabuse, apomorphine, octyl alcohol, potassium rhodanate, amphetamine, and magnesium sulfate. The results indicate that iv alcohol is the only substance which counteracts the nervous perturbations caused by alcohol given by other routes; nicotinamide and vitamins B₃ and B₄ improve the performance of iv alcohol. Apomorphine, antabuse, and octyl alcohol accelerate intoxication, and their action may lead to the production of conditional reflexes; such reactions are dangerous, and administration must be carefully supervised. Potassium rhodanate and amphetamine temporarily inhibit the nervous effects of alcoholic intoxication, but for a limited time only, and the quantities administered must be carefully graded to the degree of intoxication, since those substances not completely neutralized in such action may substitute their own toxic effects for those of alcohol.

761. Lecoq, R., Chauchard, P., and Mazoué, H.
ACTION DE QUELQUES PSYCHOTROPES DANS LE TRAITEMENT DE L'ALCOOLOMANIE. [Action of several psychotropes in the treatment of alcoholomania].
 Revue de l'Alcoolisme (Nantes), 8(4): 292-295 (0 ref.), 1962.
 F – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – nerv. syst. – barbiturates – sed., hypnot. – tranquilizers – unclass. ther. agents – *CAAAL-0 A-0869.

The effects of chlorpromazine (12.5 mg), prothipendyl (10 mg), reserpine (0.2 mg), vinylbital (10 mg), methylpentyl carbamate (25 mg), and methylpentynol carbamate (100 mg) on the nervous chronaxies of the white rat (ip administration) were studied under the following conditions: drug only, drug followed 1 hr later by injection of 0.5 mg 25% alcohol, or drug plus 5 mg disulfiram (simultaneous injection), followed by 0.5 ml 25% alcohol 1 hr later. Chlorpromazine neutralized the effects of alcohol, both with and without disulfiram. Prothipendyl alone inhibited the effects of alcohol, but, in combination with disulfiram, augmented them. Reserpine, alone or with disulfiram, did not inhibit the alcohol effect. Vinylbital alone neutralized the action of alcohol, but augmented it after disulfiram. Methylpentynol carbamate failed to influence the alcohol effect when given singly, and augmented it after disulfiram. Methylpentyl carbamate inhibited the alcohol effect in both cases.

762. Lecoq, R., Chauchard, P., and Mazoué, H.
RECHERCHES CHRONAXIMÉTRIQUES SUR L'ACTION QU'EXERCENT QUELQUES PSYCHOLEPTIQUES ET PSYCHOTONIQUES SUR LES EFFETS NERVEUX DE L'ALCOOL ÉTHYLIQUE: CONCLUSIONS PRATIQUES. [Chronaximetric investigations on the action exerted by some psycholeptics and psychotonics upon the nervous effects of ethanol: practical conclusions].
 Ann. Pharm. Franc. (Paris), 20: 607-622 (11 ref.), 1962.
 F – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – dose resp. – CNS – nerv. syst. – senses – antidepressants – barbiturates – hallucinogens – sed., hypnot. – tranquilizers – unclass. ther. agents – *CAAAL-0 A-0870.

The effects on nervous chronaxies of various hypnotics, analgesics, tranquilizers, orthoneurotics, neuroleptics, psychotonics, and psycholeptics (ip administration) were studied in white rats under the following conditions: drug alone, drug followed by 1 ml 25% alcohol ip 1 hr later, drug plus simultaneous alcohol dose after 5 days of alcohol pretreatment (1 ml 25% alcohol/day), and drug plus 0.5 ml disulfiram (simultaneous ip administration), followed by 1 ml 25% alcohol 1 hr later. It was found that the following drugs protected against the nervous effects of alcohol in the rat: gardenal (25 mg), nembutal (2.5 mg), optanox (10 mg), eunoctal (25 mg), nesdonal (20 mg), viadril (2.5 mg), dolosal (2.5 mg), butazolidin (100 mg), moderil (5 mg), librium (2.5 mg), frenquel (2.5 mg), clarmil (15 mg), statran (25 mg), largactil (12.5 mg), dominal (10 mg), nozinan (12.5 mg), phenergan (12.5 and 1.25 mg), tementil (1 mg), theralene (2.5 mg), majeptil (5 mg), melleril (5 mg), haloperidol (2.5 mg), fluanisone (1.25 mg), maxiton (5 mg), marsilid (2.5 mg), niamide (5 mg), heptamyl (2.5 mg), tofranil (6.25 mg), lucidril (20 mg), lysergamine (75 μ g), and psilocybin (3 mg). The following drugs did not afford protection against the nervous effects of alcohol: miltown (40 mg), benactyzine (20 mg), atarax (50 mg), n-oblivon (100 mg), hemineurin (10 mg), and serpasil (0.2 mg).

763. Lecoq, R., Chauchard, P., and Mazoué, H.
ÉTUDE CHRONAXIMÉTRIQUE DU COMPORTEMENT EXPÉRIMENTAL DE QUELQUES PSYCHOLEPTIQUES SUR LES EFFETS DE L'ALCOOL (AVEC ET SANS ACTION ASSOCIÉE DU DISULFIRAME). [Chronaximetric study of the experimental action of several psycholeptics on the effects of alcohol (with or without the combined action of disulfiram)].
 Académie des Sciences, Comptes Rendus Hebdomadaires des Séances (Paris), 254: 941-943 (4 ref.), 1962.
 F – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – nerv. syst. – sed., hypnot. – tranquilizers – unclass. ther. agents – *CAAAL-0 A-0871.

The effects on nervous chronaxies of 12.5 mg chlorpromazine, 40 mg procalmadiol, 25 mg methylpentanol carbamate, 100 mg methylpentynol carbamate, and 5 mg ethylmethylpentanoic acid carbamide were studied in white rats (ip administration) under the following conditions: drug alone, drug followed 1 hr later by 0.5 ml 25% alcohol, drug plus simultaneous alcohol dose after 5 days of alcohol pretreatment, and drug plus disulfiram, followed 1 hr later by 0.5 ml 25% alcohol. The results showed that chlorpromazine and methylpentanol carbamate perfectly neutralized the effects of alcohol. Ethylmethylpentanoic acid carbamide neutralized the effects of alcohol in the absence of disulfiram, but enhanced the effects in the presence of disulfiram. Methylpentynol carbamate and procalmadiol enhanced the effects of alcohol, with and without disulfiram.

764. Lecoq, R., Chauchard, P., and Mazoué, H.
ÉTUDE CHRONAXIMÉTRIQUE EXPÉRIMENTALE DE L'ACTION DE QUELQUES SÉDATIFS MINÉRAUX OU VÉGÉTAUX ET DES EFFETS ASSOCIÉS DE L'ALCOOL ÉTHYLIQUE, AVEC OU SANS DISULFIRAME. [Experimental chronaximetric study of the action of some inorganic or plant sedatives and of the combined effects of ethyl alcohol, with or

without disulfiram].

Académie des Sciences, Comptes Rendus Hebdomadaires des Séances (Paris), 257: 1403-1405 (6 ref.), 1963.

F – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – nerv. syst. – gastrointest. agents – sed., hypnot. – unclass. ther. agents – *CAAAL-10933-D2 A-0872.

Rats were injected ip with the following sedatives: chloral hydrate (5 mg), calcium bromide (5 mg), extracts of passion flower (10 mg), valerian (10 mg), or opium (5 mg). The drugs were administered with or without 5 mg disulfiram, and 0.5 ml 25% alcohol was given 60 or 90 min later. It is concluded that calcium bromide inhibits the disulfiram-alcohol effect on the chronaxies, but augments the alcohol effect with disulfiram; the plant sedatives have orthoneurotic properties reminiscent of those of real neuroleptics, and neutralize the effect of alcohol if given in large enough doses.

765. Lecoq, R., Chauchard, P., and Mazoué, H.
 ETUDE CHROMAXIMÉRIQUE EXPÉRIMENTALE DE QUELQUES AGENTS
 PSYCHOTROPES ET DE LEUR ACTION SUR LES EFFETS NERVEUX DE L'ALCOOL
 ÉTHYLIQUE. I. SÉDATIFS, ANALGÉSQUES ET HYPNOTIQUES. II.
 NEUROLEPTIQUES, TRANQUILLISANTS ET ORTHONEUROTIQUES. III.
 PSYCHOTONIQUES ET PSYCHODYSLEPTIQUES. [Experimental chronaximetric study of
 several psychotropic agents and of their action on the nervous effects of ethyl alcohol. I.
 Sedatives, analgesics and hypnotics II. Neuroleptics, tranquilizers and orthoneurotics. III.
 Psychotonics and psychodysleptics].
 Thérapie (Paris), 19: 967-989 (10 ref.), 1964.
 F – ES – SpS – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo
 – CNS – nerv. syst. – analeptics – analg., antipyret. – antidepressants – barbiturates – sed., hypnot.
 – tranquilizers – unclass. ther. agents – *CAAAL-0 A-0873.

The effects on nervous chronaxies of various hypnotics, analgesics, tranquilizers, orthoneurotics, neuroleptics, psychotonics, and psycholeptics (ip administration) were studied in white rats, under the following conditions: drug alone, drug followed by 1 ml 25% alcohol ip 1 hr later, drug plus simultaneous alcohol dose after 5 days of alcohol pretreatment (1ml 25% alcohol/day), and drug plus 0.5 ml disulfiram (simultaneous administration), followed by 1 ml 25% alcohol 1 hr later. The following are the results indicating protective (+), non-protective (-), or not definitely established (\pm) action of the various agents on the nervous effects of ethanol, alone or with disulfiram (in that order): 1-3 ml distilled lime blossom water (+,+), 10 mg passion flower extract (+,+), 10 mg valerian extract (+,+), 5 mg ammonium valerianate (+,+), 10 mg sodium bromide (-,+), 5 mg calcium bromide (-,+), 5 mg opium extract (+,+), 0.5 mg morphine hydrochloride (-,-), 100 mg butazolidin (+,-), 5 mg chloral hydrate (-,-), 2.5 mg dolosal (+,-), 2.5 mg viadril (+,-), 25 mg gardenal (+,-), 2.5 mg nembutal (+,-), 10 mg optanox (+,-), 25 mg eunocet (+,-), 20 mg nesdonal (+,-), 10 mg noludar (-,+), 10 mg doriden (-,+), 12.5 mg largactil (+,+), 10 mg dominal (+,-), 5 mg taractan (+,+), 12.5 mg nozinan (+,+), 12.5 and 1.25 mg phenergan (\pm ,+), 1 mg tementil (+,+), 2.5 mg alimemazine (+,+), 5 mg majepil (+,-), 5 mg melleril (+,-), 0.2 mg serpasil (-,-), 2.5 mg haloperidol (+,+), 1.25 mg fluanisone (+,+), 40 mg equanil (-,-), 5 mg terfluzine (+,+), 20 mg benactyzine (-,-), 2.5 mg frenquel (+,-), 15 mg clarmil (+,+), 50 mg atarax (-,-), 25 mg statran (+,+), 100 mg n-oblivon (-,-), 10 mg hemineurin (-,+), 2.5 mg librium (+,-), 5 mg moderil (+,-), 5 mg maxiton (\pm ,+), 2.5 mg marsilid (+,-), 5 mg nialamide (+,+), 2.5 mg heptamyl (+,+), 6.25 mg tofranil (+,-), 10 mg lucidril (+,+), 75 g lysergamine (+,-), and 3 mg psilocybin (+,-).

766. Lee, P. K. Y., Cho, M. H., and Dobkin, A. B.
 EFFECTS OF ALCOHOLISM, MORPHINISM, AND BARBITURATE RESISTANCE ON
 INDUCTION AND MAINTENANCE OF GENERAL ANAESTHESIA.
 Canad. Anaesth. Soc. J. (Toronto), 11(4): 354-381 (41 ref.), 1964.

E – FS – exp. cont. – exp. comp. – cross-tol. – DC (add., infra-add., unspec. incr.) – DC (unchanged)
 – mammals – acute admin. – chronic admin. – in vivo – acid-base, blood pH, elect. – cardiovasc. –
 CNS – liver, kidney – metab. proc. – nerv. syst. – respir. – anesthetics – sed., hypnot. – *CAAAL-0
 A-0874.

The responses of more than 250 rats and mice made tolerant in groups to ethanol, dihydromorphinone HC1, and methohexital were evaluated during the administration of approximately 200 individual general anesthetics, in order to identify the effects of the interaction of addicting sedative-type drugs and general anesthetics, at various stages in the development of tolerance. Ethanol tolerance was induced in male albino rats (10-20% ethanol ip or po), and, to these animals, the following anesthetics were applied: 20% diethyl ether, 0.5% methohexital (30 mg/kg ip), 1.5% methoxyflurane, 1% thiopental (30 mg/kg ip), or 0.5% methohexital (30 mg/kg ip) plus 2.5 mg/kg innovar (containing 0.5 mg/ml dehydrobenzperidol and 0.01 mg/ml phentanyl). In the ethanol-tolerant rats, induction time was "stormy" and prolonged after diethyl ether and methoxyflurane; duration of anesthesia, however, was not affected. These rats were more resistant to the onset of anesthesia after methohexital or thiopental, but methohexital-innovar did not have such an effect. It is likely that established ethanol tolerance has, in fact, no appreciable influence on the induction or maintenance response to thiopental, methohexital, or innovar.

767. Leibach, W. K.
 ZUR LEBERSCHÄDIGENDEN WIRKUNG VERSCHIEDENER ALKOHOLIKA. [Effect of
 different alcoholic beverages on liver damage].
 Deutsch. Med. Wschr. (Stuttgart), 92(6): 233-238 (41 ref.), 1967.
 G – stat. surv. – congen. stud. – drug-dep. humans – liver, kidney – *CAAAL-12322-H3
 B-0951.

The importance of congener content and beverage type in the development of liver damage was investigated in 526 alcoholics. Clinical-chemical and bioptic-histological data collected during withdrawal treatment were compared, with respect to the possible significance of beer or liquor consumption. It was found that, when the degree of functional and morphological liver damage increased, the relative frequency of exclusive beer consumption in preference to liquor did not show a statistically-significant decrease. The decrease in numbers of beer drinkers relative to liquor drinkers was, however, significantly correlated with increasing daily consumption and duration of alcohol abuse. It was concluded that the extent of demonstrable liver damage depends on the intensity of alcohol abuse, rather than on a switch by drinkers to beverages of higher alcohol and congener content. Such a switch to high alcohol and high congener liquor is a symptom of increased dependence on alcohol. The hypothesis was supported by a comparison of an exclusively beer-drinking group and an exclusively liquor-drinking group. Both groups were identical in the intensity of alcohol abuse and other relevant parameters, and were identical in the extent of liver damage. It is concluded that ethanol is the decisive factor in the development of liver damage, and that congener content of the beverage consumed is only of secondary importance.

768. Leibach, W. K.
 LIVER CELL NECROSIS IN RATS AFTER PROLONGED INGESTION UNDER THE
 INFLUENCE OF AN ALCOHOL-DEHYDROGENASE INHIBITOR.
 Experientia (Basel), 25(8): 816-818 (12 ref.), 1969.
 E – GS – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin.
 – in vivo – blood lev. – liver, kidney – metab. proc. – *CAAAL-0 B-0952.

The in vivo inhibition of ethanol oxidation by pyrazole was studied in 4 groups of 11 non-fasted male Wistar rats. The blood alcohol concentration (BAC) was determined 24 hr after administration of a single dose of 6.4 g/kg ethanol po, alone or in combination with 32, 50, or 70 g/kg pyrazole po; in the order of the preceding administrations, the BAC was found to be 2, 28, 39, and 72 mg/100

ml, indicating an apparent dose-dependent inhibition. In another experiment, rats fed for 5 weeks on a 15% ethanol sol as sole source of drinking fluid were given a threshold dose of 31 mg/kg pyrazole/-day po. Death followed after 5-19 days, and liver examinations revealed a massive liver cell necrosis, with inflammatory cell reaction and fatty degeneration of surviving parenchyma. Studies of livers of rats given a single po dose of 6.0-7.7 g/kg ethanol plus 36.6 mg/kg pyrazole/day until death (less than 1 week to 68 days) revealed an effect similar to frank hepatotoxic agents in more than half of the animals. It is considered probable that the pyrazole-induced inhibition of ethanol metabolism increased ethanol toxicity, resulting in liver cell damage of a severity hitherto not reported with conventional feeding techniques, although ethanol may have also enhanced pyrazole toxicity.

769. Leloir, L. F., and Muñoz, J. M.
 ETHYL ALCOHOL METABOLISM IN ANIMAL TISSUES.
 Biochem. J. (London), 32: 299-307 (22 ref.), 1938.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged)
 – mammals – acute admin. – in vitro – G.I. tract – liver, kidney – metab. proc. – coagulants – elect.,
 water-bal. agents – miscellaneous – nutritive agents – unclass. ther. agents – *CAAAL-934-A2
 A-0875.

Ethanol oxidation was studied in rat and pigeon tissue slices. Aerobically, the rate of oxidation was found to be high in rat liver, and low in pigeon liver. In other organs, except kidney and pigeon muscle, oxidation was very low or absent. Pyruvic, lactic, succinic, fumaric, malic, and oxaloacetic acids, and alanine increased aerobic alcohol disappearance. Acetic, butyric, β -hydroxybutyric, and ascorbic acids, glycine, ornithine, glucose, ammonium chloride, and insulin showed no action, while glycerol slightly depressed alcohol oxidation. The effects of various inhibitors were also studied. Cyanide greatly decreased alcohol oxidation, but did not stop it completely. Iodoacetate, fluoride, arsenate, phloridzin, malonate, and oxalate also decreased alcohol oxidation. 2,4-dinitrophenol increased oxidation at 1.1 times 10^{-5} M concentration, and decreased it at higher concentrations.

770. Lendle, L.
 SERIENVERGIFTUNG DURCH DEN GENUSS EINES GEMISCHES VON
 TETRACHLORKOHLSTOFF UND ÄTHYLALKOHOL. [Mass poisoning by ingestion of a
 mixture of carbon tetrachloride and ethyl alcohol].
 Sammlung von Vergiftungsfällen (Berlin), 12(9): 123-124 (8 ref.), 1942.
 G – SEC – general – DC (add., infra-add., unspec. incr.) – humans – G.I. tract – liver, kidney –
 anti-infectants – *CAAAL-0 A-0876.

The author reports on a case of carbon tetrachloride mass poisoning which occurred in 1941. 13 Russians drank a mixture of alcohol and carbon tetrachloride—some drank only a few tablespoonfuls. Of this group, 9 died within 2 hr; the remaining 4 lost most symptoms of the poisoning within 4 days, although they still complained of a feeling of nausea. There were, evidently, some symptoms pointing to hepatic damage, since Lendle states that the victims began to recover from their poisoning after 10-14 days, at which time there were no longer any symptoms of such damage.

771. Leonard, B. E., and Wiseman, B. D.
 THE EFFECT OF ETHANOL AND AMPHETAMINE MIXTURES ON THE ACTIVITY
 OF RATS IN A Y-MAZE.
 J. Pharm. Pharmacol. (London), 22(12): 967-968 (3 ref.), 1970.
 E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – CNS –
 amphetamines – *CAAAL-0 B-1014.

To determine whether ethanol has the ability, like that of sodium amylobarbitone, to potentiate the effect of amphetamine, 20 male rats were divided into 4 groups of 5 animals. Each animal was run

through a Y-maze once a week. For 1 week no drug was given; in the remaining weeks, the rats were given the following compounds (in a vol of less than 0.5 ml/rat) ip prior to being run in the maze: saline sol (0.5 ml), ethanol (50-800 mg/kg), amphetamine (4 mg/kg), or ethanol + amphetamine (various amounts of ethanol + 4 mg/kg amphetamine). The lowest dose of ethanol given alone slightly increased maze activity, but higher doses decreased exploratory activity. Amphetamine increased activity. With the combined dose, it was apparent that the depressant effect of ethanol was antagonized by amphetamine, but at no dose combination did the activity of the animals approach that of amphetamine alone. It is concluded that ethanol has a different effect on amphetamine-pretreated rats from that of sodium amylobarbitone. This suggests that amylobarbitone potentiation of amphetamine is probably a specific mechanism which may not be shared by other drugs.

772. Leonards, J. R.
FAECAL BLOOD-LOSS AFTER SODIUM ACETYLSALICYLATE TAKEN WITH ALCOHOL.
 Lancet (London), 1: 943 (3 ref.), 1969.
 E – exp. – general – DC (unchanged) – humans – cardiovasc. – G.I. tract – analg., antipyret. – *CAAAL-13819 B-0535.

The author comments on the significance of data in the experimental study by Bouchier and Williams (Lancet, 1(7587): 178-180, 1969), concerning faecal blood-loss after combined sodium acetylsalicylate and alcohol ingestion. In 19 subjects in this study, the mean faecal blood-loss was 0.36 ml/day for the control, 0.45 ml/day after alcohol plus sodium acetylsalicylate, and 0.40 ml/day after alcohol plus a placebo. The mean average difference in faecal blood-loss of 0.05 ml/day between the drug and placebo group was within the range of experimental error for the method. The mean blood-loss in the drug period was well below the upper limit of normal daily faecal blood loss (which is up to 1.5 ml/day). In his own unpublished experimental work, the author reports that large amounts of 45% alcohol and sodium acetylsalicylate did not increase the average faecal blood-loss above the control value of 0.7 ml/day. It is concluded that faecal blood-loss cannot be imputed to sodium acetylsalicylate taken with alcohol.

773. Lerner, J.
SIDE REACTIONS OF THE PHENOTHIAZINES AND THE THERAPEUTIC USE OF ETHYL ALCOHOL.
 Amer. J. Psychiat. (Hanover), 121(9): 919-920 (6 ref.), 1965.
 E – general – DC (antidotal) – psychot. humans – CNS – skel., muscle, skin – autonomic agents – tranquilizers – *CAAAL-11451-V1 B-0354.

A case is reported of a woman who was hospitalized because of a schizophrenic condition. She was treated with fluphenazine dihydrochloride (2.5 mg/tablet, 5 tablets in 2 1/2 days), which resulted in severe dystonic movements. The patient's vital signs were entirely normal, but she was so hyperactive that she had to be placed in seclusion. The tongue protruded involuntarily, and was almost continuously extended, speech was extremely dysarthric, there were dystonic movements of the head, the eyes turned upwards and outwards for short intervals, and the extremities were markedly stiff. 2.5 mg kemadrin was twice administered without effect. 2 doses of whiskey (2 oz/dose), 15 min apart, were then given, and, within 2 hr, there was a complete cessation of all symptoms. The author concludes that ethanol can rapidly ameliorate complications resulting from the use of some phenothiazine derivatives.

774. Lester, D.
THE ACETYLATION OF ISONIAZID IN ALCOHOLICS.
 Quart. J. Stud. Alcohol (New Haven), 25(3): 541-543 (6 ref.), 1964.
 E – SEC – exp. cont. – cross-tol. – humans – drug-dep. humans – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – cardiovasc. – anti-infectants – *CAAAL-10447-C29 A-1301.

A study of 28 male alcoholics was conducted, to investigate if the rate of isoniazid acetylation in alcoholics differs from that in non-alcoholics, and whether this is due to the enzyme level or the acetate availability. The half-life of isoniazid and its subsequent bimodal distribution were determined. Analysis of the various drinking-pattern types with the isoniazid phenotype was not informative. To rule out any other factors which could cause disparate isoniazid rates, 2 non-alcoholics were tested, and found to have widespread differences in isoniazid rates, the half-life being 250 min in one, and 110 min in the other. With no plasma-binding ability of isoniazid determined, nor any apparent deacetylase influence on the rate difference, the maintenance of 0.02 to 0.04% blood alcohol before and during half-life determination produced a 30% decrease in the half-life of isoniazid in both subjects. The results offer no proof for the hypothesis that biological reasons for the use of alcohol should be sought in its chemical nature and manner of disposal, especially its oxidative metabolism to acetate. However, reactions other than the acetylation of exogenous aryl amines may be dependent to a greater extent on acetate availability and rate of utilization.

775. Lester, D.

FACTORS INFLUENCING THE METABOLISM AND DISAPPEARANCE OF ALCOHOL. In: Harger, Rolla N., ed. *Alcohol and Traffic Safety*. Proceedings of the Fourth International Conference on Alcohol and Traffic Safety at Indiana University, December 6-10, 1965. Bloomington, Indiana: Indiana University Press, pp. 267-274 (63 ref.), 1966.

E – presentation – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – mammals – in vivo – in vitro – liver, kidney – metab. proc. – elect., water-bal. agents – hormones, hormone antag. – nutritive agents – *CAAAL-0 B-0355.

The literature is reviewed from the point of view of forensic science. The influence of sleep, unconsciousness, heavy muscular work, increased losses in respiration and perspiration, fasting and feeding, and the influence of most of the drugs thus far investigated, on the rate of disappearance of alcohol as studied in human and animal experiments, are discussed. Only fructose seems to influence the oxidation of alcohol significantly, and even for this agent there are great disadvantages.

776. Lester, D., Keokosky, W. Z., and Felzenberg, F.

EFFECT OF PYRAZOLES AND OTHER COMPOUNDS ON ALCOHOL METABOLISM.

Quart. J. Stud. Alcohol (New Haven), 29(2): 449-454 (15 ref.), 1968.

E – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – dose resp. – blood lev. – liver, kidney – metab. proc. – alcohols – analg., antipyret. – *CAAAL-12478-A2 B-0356.

Groups of 2-20 male rats were injected ip with possible inhibitors and non-inhibitors of alcohol metabolism, followed by 0.5-1.0 g alcohol (7.5% w/v)/kg, and the blood alcohol concentration was determined by gas chromatography. The following compounds, in doses of less than 0.5 mmole/kg, inhibited the rate of alcohol disappearance by 25% or more from a mean rate of 0.330 ± 0.34 g/kg/hr: pyrazole, 4-bromopyrazole, 4-methylpyrazole, 4-ethylpyrazole, 4-decylpyrazole, 4-carbethoxypyrazole, 4-(β -hydroxyethyl) pyrazole, 4-(γ -hydroxypropyl) pyrazole, 4-(γ -chloropropyl) pyrazole, 4-(γ -aminopropyl) pyrazole, o-phenanthroline, and 2,2'-bipyridyl. In doses of 0.37-1.32 mmole/kg, the following did not inhibit the disappearance of alcohol: 3-methylpyrazole, 3,4-dimethylpyrazole, 3,5-dimethylpyrazole, 4-amyl-3-methylpyrazole, 1-methylol-3-undecylpyrazole, 3,5-pyrazoledicarboxylic acid, 3-methyl-2-pyrazolin-5-one, antipyrine, 2,5-dimethylpyrrole, and 2-methylpyrazine. 3.40 mmole of n-butoxyethanol, 40 mmole of dihydroxyacetone, and 60 mmole of propylene glycol also failed to inhibit disappearance.

777. Lester, D., and Benson, G. D.

PYRAZOLE INHIBITION OF IN VIVO ALCOHOL OXIDATION.

Fed. Proc. (Bethesda), 28(2): 546 (0 ref.),

1969.

E – abst. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – liver, kidney – metab. proc. – *CAAAL-0 B-0357.

Pyrazole inhibition of in vivo methanol-1-¹⁴C, and butanol-1-¹⁴C oxidation was studied in rats. Respiratory CO₂ was collected for 6 hr. Methanol (1.0 g/kg), ethanol (1.0 g/kg), and butanol (0.5 g/kg) were recovered in the form of CO₂ as 17, 89, and 81% of the dose. Pyrazole (150 mg/kg), given 30 min before the alcohols, inhibited methanol, ethanol, and butanol oxidation by 57%, 73%, and 41%, respectively. Gas liquid chromatography determination of ethanol and butanol showed an inhibition of 93 and 43%.

778. Leuschner, F., and Leuschner, A.
 VERGLEICHENDE TIEREXPERIMENTELLE UNTERSUCHUNGEN MIT WEINEN AUS HYBRIDEN- UND EUROPÄERREBEN. [Comparison of wines of hybrid and pure European origin in animal experiments].
 Arzneimittelforschung (Aulendorf), 17: 59-66 (24 ref.), 1967.
 G – ES – exp. cont. – exp. comp. – congen. stud. – mammals – chronic admin. – in vivo – dose resp. – liver, kidney – *CAAAL-0 B-0953.

The long-term effects of hybrid wine (from grapes developed from a crossing of European and native American varieties), pure wine (from grapes of pure European origin), and a mixture of various alcohols corresponding to the alcohol concentrations contained in the wines, were compared in male and female Wistar rats (70-110 g). The ED₅₀ values for pure wine, hybrid wine, and the alcohol mixture were established at 15.2, 15.5, and 14.4 ml/kg, respectively. Over a 4-month period, each test sol was administered in doses of 20, 40, or 80 ml/kg/day to both male and female rat groups. In another experiment, the same dosages were administered for 4 weeks to rats which were also given Handler's diet to induce moderate fatty liver. The development of body wt and the increase of total hepatic lipid content were found to be influenced by all 3 sol to approximately the same extent. Histological changes of liver tissue were observed with both wines, but not with the alcohol mixture. High doses (80 ml/kg/day) of the alcohol mixture aggravated experimentally-induced fatty degeneration of the liver, whereas only low doses (20 and 40 ml/kg/day) of both wines had a similar effect. It is concluded that tolerance to both wines is lower than to the alcohol mixture, and that the observed differences in several test criteria between the wines and the alcohol mixture can be attributed to the congener content of the former. Neither test series yielded evidence of a difference in the effects of hybrid and pure wines.

779. Levy, G., Miller, K. E., and Reuning, R. H.
 EFFECT OF COMPLEX FORMATION ON DRUG ABSORPTION. III. CONCENTRATION- AND DRUG-DEPENDENT EFFECT OF A NONIONIC SURFACTANT.
 J. Pharm. Sci. (Washington), 55(4): 394-398 (15 ref.), 1966.
 E – exp. cont. – DC (unchanged) – other org. – chronic admin. – in vivo – other drug lev. – absorp., distrib., stor. – *CAAAL-0 B-0954.

The effects of various concentrations of the non-ionic surfactant, polysorbate 80 (P 80) on the absorption of a number of alcohols and barbiturates were studied in goldfish. P 80 had no effect on the rate of ethanol absorption, as judged by time of death after immersion in 5% ethanol with up to 2.0% surfactant. Equilibrium dialysis showed that there was no binding of ethanol by P 80. In contrast to this, low concentrations of P 80 increased, and high concentrations decreased, secobarbital absorption. In attempting to discover the reason for the different effects of P 80 on ethanol and secobarbital absorption, the possibility that ethanol itself decreased the surface tension so much that P 80 had no effect was ruled out experimentally. The answer may lie in different routes of absorption of the drugs—ethanol diffuses across membranes through pores, whereas secobarbital diffuses through the lipid barrier. P 80 had no effect on absorption of other alcohols of low molecular wt,

but significantly increased pentobarbital absorption. It is concluded that the absorption-enhancing action of P 80 may be due either to the formation of a non-micellular drug-surfactant complex, or to a direct action of the surfactant on the lipoid barrier of the cell membrane.

780. Lewis, D. J., and McIntire, R.

A CONTROL FOR THE DIRECT MANIPULATION OF THE FRACTIONAL ANTICIPATORY GOAL RESPONSE.

Psychol. Rep. (Missoula), 5: 753-756 (1 ref.),

1959.

E – exp. cont. – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – mot. perform.
– anesthetics – *CAAAL-9622-J2 A-0877.

Activity wheel revolutions were recorded with 12 rats for 1/2 hr before, and for 1/2 hr under the following drug conditions: .20 cc pilocarpine in water, .20 cc benzocaine in 64% alcohol, .20 cc 64% alcohol, .20 cc water, and a sham administration. All drugs were administered po. None of the treatments had a differential effect on activity, and the results tend to support the idea that differential effects of pilocarpine and benzocaine are due to their effect on the fractional anticipatory goal response, and not to any differential effects on activity.

781. Lewis, W., and Schwartz, L.

AN OCCUPATIONAL AGENT (N-BUTYRALDOXIME) CAUSING REACTION TO ALCOHOL.

Med. Ann. D.C. (Washington), 25: 485-490 (13 ref.),

1956.

E – general – DC (sensit.) – humans – cardiovasc. – skel., muscle, skin – *CAAAL-7620-C3

A-1303.

A sensitization reaction to alcohol produced by the antioxidant n-butylaldoxime is described. It was noticed by workers in a large printing factory that, if they consumed alcohol after work, they would experience an unpleasant reaction, characterized by a reddish, virescent, non-itching, coalescing, macular rash of the face, neck and upper trunk, congestion of the conjunctiva and nares, and congestion of the mucous membranes of the throat, which caused dyspnea without wheezing, and palpitation. The symptoms commenced sometimes within 10 min after consumption of as little as 6 oz of beer, and usually lasted about 1-2 hr; their severity was directly proportional to the amount of alcohol consumed. Investigation revealed that the workers were being sensitized by absorbing an anti-skinning compound (n-butylaldoxime) as it evaporated from the ink in the presses. Acetaldehyde blood levels were found to be elevated by the absorption of n-butylaldoxime, and elevated still further after the ingestion of alcohol. It is concluded that n-butylaldoxime causes a sensitization reaction to alcohol similar to, but milder than, the antabuse-alcohol reaction.

782. Lewis, W. B.

CAFFEINE, IN ITS RELATIONSHIPS TO ANIMAL HEAT AND AS CONTRASTED WITH ALCOHOL.

Brit. J. Psychiat. (London), 29: 167-178 (0 ref.),

1883.

E – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – cardiovasc. – metab. proc.
– skel., muscle, skin – stimulants – *CAAAL-0 A-1302.

The author performed a series of experiments on the influence of caffeine on the body temperature of rabbits. In 1 experiment, rabbits received, on separate occasions, caffeine, alcohol, or 6 grains of caffeine in combination with 2 drachms of alcohol. It was found that, whereas in the normal state 1.08 heat units is the maximum heat loss attained, the maximum was 4.62 after caffeine and 4.28 after alcohol, the stage of increased thermogenesis after both substances lasting for more than 1 hr. On the other hand, the initial heat loss after caffeine was succeeded by a retention of heat and increase of temperature, whereas, after alcohol, the heat loss was continuous and prolonged. After the com-

bined dose, however, an early stage of diminished heat-formation preceded the increased thermogenesis, but the fall in body temperature normally produced by alcohol was completely antagonized by caffeine. The author advocates the therapeutic and dietetic use of caffeine as an antagonist of alcohol, and suggests the use of coffee and tea in place of alcohol whenever retention of body heat is essential.

783. Lickint, F.

UEBER DIE AUSLÖSUNG ABNORMER ALKOHOL-REAKTIONEN DURCH MEDIKAMENTE (ZUGLEICH EINE WARNUNG FÜR VERKEHRSTEILNEHMER). [On the precipitation of abnormal alcohol reactions by drugs, and a warning to participants in traffic].

Suchtgefahren (Hamburg), 1(4): 1-9 (0 ref.),

1956.

G – general – review – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – mammals – absorp., distrib., stor. – cardiovasc. – CNS – G.I. tract – metab. proc. – analg., antipyret. – anti-infectants – barbiturates – sed., hypnot. – *CAAAL-8126-D3 A-0878.

The author reviews current knowledge on the subject. Some drugs intensify the noxious effects of alcohol. Isoniazid produced severe intoxication in a human, 1 hr after ingestion of 1 bottle of beer, and other physicians report similar cases, including 1 death. Experiments with rabbits confirmed that isoniazid reduces alcohol tolerance. Amidopyrine has produced a disulfiram-like reaction at blood alcohol levels of 70 to 110 mg%. Irgapyrin (amidopyrine + phenylbutazone) also precipitates an abnormal alcohol reaction. Barbiturates and alcohol have a mutual potentiative action. Tobacco intensifies the effect of alcohol. Certain mushrooms (*Coprinus atramentarius*), agricultural chemicals (e.g., cyanamide), and sulfonamide-containing compounds can also cause unfavourable reactions with alcohol. No drug is known which can effectively counteract the impairment of driving ability caused by alcohol.

784. Lickint, F.

DIE AUSLÖSUNG ABNORMER ALKOHOLREAKTIONEN DURCH MEDIKAMENTE.

[The evoking of abnormal alcohol reactions by drugs].

Therapiewoche (Karlsruhe), 7(13): 414-418 (0 ref.),

1957.

G – general – review – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – mammals – absorp., distrib., stor. – cardiovasc. – CNS – G.I. tract – metab. proc. – analg., antipyret. – anti-infectants – barbiturates – cardiovasc. agents – sed., hypnot. – *CAAAL-8126-D3 A-0879.

The author reviews current knowledge on the subject. Some drugs intensify the noxious effects of alcohol. Isoniazid produced severe intoxication in a human, 1 hr after ingestion of 1 bottle of beer, and other physicians report similar cases, including 1 death. Experiments with rabbits confirmed that isoniazid reduces alcohol tolerance. Amidopyrine has produced a disulfiram-like reaction at blood alcohol levels of 70 to 110 mg%. Irgapyrin (amidopyrine + phenylbutazone) precipitates an abnormal alcohol reaction. Barbiturates and alcohol have a mutual potentiative action. Tobacco intensifies the effect of alcohol. Certain mushrooms (*Coprinus atramentarius*), agricultural chemicals (e.g., cyanamide), and sulfonamide-containing compounds can also cause unfavourable reactions with alcohol. No drug is known which can effectively counteract the impairment of driving ability caused by alcohol.

785. Lickint, F.

KANN DIE ALKOHOLWIRKUNG DURCH MEDIKAMENTE GESTEIGERT WERDEN?

[Can the effect of alcohol be increased by drugs?].

Deutsch. Gesundh. (Berlin), 14(15): 665-672 (0 ref.),

1959.

G – RS – general – review – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – mammals – metab. proc. – analg., antipyret. – anti-infectants – barbiturates – cardiovasc. agents – gastrointest. agents – sed., hypnot. – *CAAAL-9311-D3 A-0880.

The synergistic or sensitizing effects of various drugs in combination with alcohol are reviewed, and the possible mechanisms outlined. Discussed are: isonicotinic acid hydrazide, pyramidon, barbiturates, urea derivatives, polamidone compounds, salicylates, rhodan compounds, sulfonamides, ataractics, and antihistamines. The implications for driving impairment are pointed out, and an appeal is made to the pharmaceutical industry to include a warning against alcohol intake on labels of products.

786. Lieber, C. S., and Rubin, E.

ETHANOL—A HEPATOTOXIC DRUG.

Gastroenterology (Baltimore), 54(4): 642-646 (45 ref.),

1968.

E – review – humans – drug-dep. humans – mammals – liver, kidney – metab. proc. – *CAAAL-12899-B1 B-0955.

The classification of ethanol among the hepatotoxic agents, its role as an inducer of drug-metabolizing enzymes in the liver, and the implications of the induction of hepatic drug-detoxifying enzymes by ethanol, are discussed. It is concluded that the fact that ethanol induces proliferation of the smooth endoplasmic reticulum sheds a new light on a number of thus far unexplained effects of alcohol in the field of drug interaction, and in lipid, and, possibly, porphyrin, metabolism. In addition to these changes which facilitate drug detoxication (and can therefore be considered adaptive), there are many other aspects of ethanol hepatotoxicity, including marked alterations in mitochondria, which may represent, in part, the morphological counterpart of the notable decrease, after ethanol administration, in the capacity of isolated livers, liver slices, and mitochondria to oxidize various substrates, including lipids. The theory that ethanol acts as a hydrogen donor can explain a number of its effects, while other changes can be attributed to its metabolites, acetaldehyde and acetate.

787. Lieber, C. S., and DeCarli, L. M.

EFFECT OF DRUG ADMINISTRATION ON THE ACTIVITY OF THE HEPATIC MICROSOMAL ETHANOL OXIDIZING SYSTEM.

Life Sci. (Oxford), 9(2): 267-276 (23 ref.),

1970.

E – exp. cont. – exp. comp. – cross-tol. – mammals – acute admin. – chronic admin. – in vivo – blood lev. – species or sex diff. – liver, kidney – metab. proc. – barbiturates – *CAAAL-13958 B-0536.

The effects of 3-amino-1,2,4-triazole (AT), butylated hydroxytoluene (BHT), phenobarbital (Pb) and 3-methylcholanthrene (MC) on the activity of the microsomal ethanol-oxidizing system (MEOS) was studied in rats. 1 group of rats was fed a nutritionally adequate liquid diet for a 24-day period, containing 36% of the total calories as ethanol (16.5 ± 0.33 g/kg per day); a control group received an equivalent amount of carbohydrate. Paired rats of the ethanol-pretreatment and control groups received the following drug administrations: AT (1 g/kg, ip), BHT (0.84 ± 0.06 g/kg per day for 6 days, po), sodium Pb (80 mg/kg daily for 4 days, ip), or MC (20 mg/kg, ip). MEOS activity increased in all groups, but most strikingly after ethanol, whether expressed per g of liver, per mg of microsomal protein, or per 100 g of body wt. MEOS activity, expressed per mg of microsomal protein, did not increase significantly after administration of other drugs. It is concluded that MEOS activity is less affected by other drugs than it is by ethanol feeding.

788. Lieber, C. S., and DeCarli, L. M.

HEPATIC MICROSOMAL ETHANOL-OXIDIZING SYSTEM: *IN VITRO* CHARACTERISTICS AND ADAPTIVE PROPERTIES *IN VIVO*.

J. Biol. Chem. (Baltimore), 245(10): 2505-2512 (45 ref.),

1970.

E – exp. cont. – mammals – acute admin. – chronic admin. – in vivo – in vitro – blood lev. – species or sex diff. – liver, kidney – metab. proc. – cardiovasc. agents – unclass. ther. agents – *CAAAL-14486 B-1015.

Sprague-Dawley rats were pair-fed liquid diets containing ethanol or isocaloric carbohydrate, and were decapitated after various periods; some animals received pyrazole (4.4 mmoles/kg po), 3-amino-

1,2,4-triazole (AT—1 g/kg ip), or an isotonic saline sol, prior to decapitation. Other animals received a non-liquid diet, plus acute administrations of liquid diet and pyrazole, or of ethanol or glucose, prior to killing. Blood clearances were based on tail vein sampling. Inhibitors—carbon monoxide (40%), sodium cyanide (0.1 mM), pyrazole (2 and 4 mM), sodium azide (0.1 mM), and SKF-525-A (1 mM)—were studied in vitro in rat liver slices. At physiological pH (optimum), substantial NADPH-dependent ethanol oxidation occurred in the microsomal fraction only; in contrast, alcohol dehydrogenase activity was localized in the cytosol, and was optimum at pH 10-11. Replacement of air by nitrogen almost abolished ethanol oxidation, and carbon monoxide and sodium cyanide inhibited it. Cyanide, pyrazole, azide, and AT partially or completely failed to inhibit the NADH-dependent microsomal ethanol oxidation system under conditions which diminished catalase activity, while activity of alcohol dehydrogenase in cytosol and microsomal and total hepatic catalase did not increase. Pyrazole reduced, but did not block, ethanol metabolism in vivo or in liver slices. Even after pyrazole, ethanol clearance rates remained higher in ethanol-pretreated rats.

789. Lieber, C. S., Rubin, E., and DeCarli, L. M.
 HEPATIC MICROSOMAL ETHANOL OXIDIZING SYSTEM (MEOS):
 DIFFERENTIATION FROM ALCOHOL DEHYDROGENASE AND NADPH OXIDASE.
 Biochem. Biophys. Res. Commun. (New York), 40(4): 858-865 (23 ref.), 1970.
 E – exp. cont. – exp. comp. – mammals – acute admin. – in vitro – liver, kidney – metab. proc. –
 analg., antipyret. – *CAAAL-0 B-0956.

The relationship of the hepatic microsomal ethanol oxidizing system (MEOS) to alcohol dehydrogenase (ADH) and NADPH oxidase was investigated in rat livers. Hepatic cytosol and washed microsomes were prepared, and the ADH, NADPH and MEOS activities were measured in both fractions. The effects of pyrazole (1, 2, and 4 mM) and dimethylsulfoxide (DMSO—10, 50, and 100 mM) on ADH, and the influence of these compounds and of sodium cholate (14 mM) on the activity of MEOS, NADPH oxidase, aniline hydroxylase, and aminopyrine demethylase were then studied. The effect of sodium cholate on microsomal cytochrome P₄₅₀ was also determined. 2 and 4 mM pyrazole abolished ADH activity, but reduced MEOS activity by only 11% and 22%, respectively. 1 mM pyrazole decreased ADH activity by 98%, but had no significant effect on MEOS activity. No sign of reduction of MEOS activity by DMSO was found, whereas 100 mM DMSO reduced ADH activity by 80%. Cholate inhibited NADPH oxidase, as well as other microsomal enzymes, by 80%, and also inactivated cytochrome P₄₅₀. The results support the hypothesis that ADH plays no role in the MEOS, and no evidence was found to link MEOS activity with that of microsomal NADPH oxidase.

790. Lieber, C. S.
 INTERACTIONS OF ETHANOL, DRUG AND LIPID METABOLISM; ADAPTIVE
 CHANGES AFTER ETHANOL CONSUMPTION.
 Clin. Sci. (London), 39: 8P-9P (4 ref.), 1970.
 E – abst. – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals –
 liver, kidney – metab. proc. – unclass. ther. agents – *CAAAL-0 B-0957.

Chronic ethanol feeding, in man and rat, produced a proliferation of the smooth endoplasmic reticulum (SER), a doubling of the microsomal ethanol oxidizing system (MEOS) activity, and enhanced rates of blood ethanol clearance. The alcohol dehydrogenase (ADH) did not increase. There were also increased hepatic lipoprotein production, enhanced activities of other hepatic microsomal drug detoxifying enzymes, and accelerated blood clearance of various drugs. In contrast, acute ethanol administration retarded drug clearance, and, in vitro, ethanol inhibited microsomal drug detoxification. Thus, it is concluded that the adaptive response of MEOS helps to explain various ethanol effects, including SER proliferation, induction and inhibition of hepatic microsomal drug detoxifying enzymes, the interactions of ethanol, drug and lipid metabolism, and the metabolic tolerance to ethanol in alcoholics.

791. Linck, K.

HINWEISE AUF ENTSTEHUNG, VERHÜTUNG UND BEHANDLUNG DER AKUTEN ALKOHOLVERGIFTUNG. [Hints on the genesis, prevention and treatment of acute alcohol poisoning].

Med. Klin. (Munich), 44(29): 931-934 (27 ref.),

1949.

G – general – DC (antidotal) – humans – blood comp., sites, lymph – CNS – respir. – autonomic agents – cardiovasc. agents – stimulants – *CAAAL-5286-N7 A-0881.

The author distinguishes intoxication and poisoning “in the narrow sense” (such poisoning begins with slurred speech and uncertain gait, and can develop into cyanosis, coma, convulsions, respiratory paralysis, and death). Whereas the intoxicated person can be aroused and will react to stimuli, the poisoned person does not so react, and requires medical aid. Recommended treatment for poisoning includes: stomach lavage, artificial respiration using 90% oxygen and 10% CO₂, administration of coramine, and, also, cardiazole, caffeine, ephedrine, digitalis, strychnine, lobeline, or (in some cases) nitroglycerin may be used to advantage. Skin stimulation and external heat application should be maintained. All alcohol poisoning cases can be saved by prompt application of these measures.

792. Lind, N., and Parkes, M. W.

EFFECTS OF INHIBITION AND INDUCTION OF THE LIVER MICROSOMAL ENZYME SYSTEM ON THE NARCOTIC ACTIVITY OF ETHANOL IN MICE.

J. Pharm. Pharmacol. (London), 19: 56-57 (6 ref.),

1967.

E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – CNS – liver, kidney – antidepressants – barbiturates – tranquilizers – *CAAAL-11885-B2 B-0358.

The authors investigated the possibility that drugs known to affect the liver microsomal enzyme system, either by inhibiting or inducing activity, might thereby alter the pharmacological effectiveness of ethanol. Groups of 10 female mice were used for each test. Pretreatment with 25 mg/kg β -diethylaminoethyldiphenylpropyl acetate (SKF 525A) ip, 45 min before 4.5 or 5.0 g/kg ethanol ip (20% v/v sol in water), prolonged ethanol sleeping time fourfold; this was associated with a potency increase of 1.1 (1.083-1.139). Pretreatment with 2 mg/kg chlorpromazine, 50 mg/kg pentobarbitone, 10 or 20 mg/kg amitriptyline, and 40 mg/kg imipramine (ip administrations), 21 hr before ethanol (5.5-5.75 g/kg ip, 20% v/v sol in water), had the following effects: chlorpromazine and pentobarbitone considerably shortened sleeping time, and 20 mg/kg amitriptyline also shortened sleeping time; imipramine did not cause any significant effect.

793. Lindsly, H.

ALCOHOL AS A REMEDY FOR THE POISON OF THE RATTLESNAKE.

Stethoscope and Virginia Medical Gazette (Richmond), 2: 540-541 (0 ref.),

1852.

E – general – case hist. – DC (antidotal) – humans – *CAAAL-0

A-0882.

The author describes a case of rattlesnake bite which he encountered. The victim, a soldier, was bitten on the hand, and given a large quantity of brandy (more than a pint) by his friends, who then brought him to the author for treatment. The patient was found too intoxicated to be roused, so he was sent back to his army quarters. The man was very ill during the night from the effects of the brandy, but subsequently experienced no ill effects from the poisoning.

794. Linke, H.

PSYCHOPHARMAKA IN DER INNEREN MEDIZIN, UNTER BESONDERER BERÜCKSICHTIGUNG IHRER EINFLUSSNAHME AUF DIE

VERKEHRSTÜCHTIGKEIT. [Psychopharmacological agents in internal medicine with particular consideration of their influence on efficiency in traffic].

Deutsch. Med. J. (Berlin), 17(20): 591-597 (0 ref.), 1966.
 G – SEC – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – CNS – metab. proc.
 – *CAAAL-0 B-0359.

The therapeutic applications and effects of psychopharmacological drugs are classified, and the specific properties of phenothiazine derivatives are tabulated. Reference is made to possible potentiation when alcohol is combined with neuroleptics, a potentiation which is brought about by toxic substances in the intermediate metabolism on the one hand, and actual synergism between alcohol and drugs on the other. It is further stated that synergism between insignificant blood alcohol concentrations and subliminal doses of psychopharmacological drugs impairs the driving ability of the motorist, due to ensuing intoxication.

795. Lisboa, P. E., Castel-Branco, N., and Sá Marques, M. M.
 ANOREXIA FOR ALCOHOL: A SIDE-EFFECT OF PHENETHYLBIGUANIDE.
 Lancet (London), 1: 678 (5 ref.), 1961.
 E – general – case hist. – DC (sensit.) – humans – cardiovasc. – glands – anti-infectants –
 *CAAAL-9185-E3 A-1339.

An unexpected finding during trial use of phenformin as an oral antidiabetic agent was the loss of desire and even repugnance for alcohol, associated in some cases with a metallic taste after ingestion of alcoholic beverages. Of 50 cases, comprising 17 abstainers and 33 regular drinkers, 10 showed this side effect. The effect was noticed during the first days of treatment with relatively low doses of phenformin (75-100 mg/day), but it is considered probable that the frequency and intensity of the reaction is roughly proportional to the dose. Case histories of 1 chronic alcoholic and 2 moderate drinkers are presented. This subjective effect can only be assessed by the double-blind technique, but, with further research, phenformin may be found useful in treatment of alcoholism, especially since it appears that healthy persons are resistant to the hypoglycemic effect of this agent.

796. Lisboa, P. E., Castel-Branco, N., and Sá Marques, M. M.
 ANOREXIA PARA BEBIDAS ALCOÓLICAS, UM EFEITO AINDA NÃO DESCRITO DA
 ADMINISTRAÇÃO DE FENFORMINA. [Anorexia for alcoholic beverages: an effect not yet
 described of phenformin administration].
 Jornal do Medico (Oporto), 44: 113-116 (5 ref.), 1961.
 P – E – general – case hist. – DC (sensit.) – humans – in vivo – psychol. perform. – cardiovasc. –
 glands – hormones, hormone antag. – *CAAAL-9185-E3 A-1340.

A side effect of the oral antidiabetic drug phenformin (phenethylbiguanide) has been observed during therapeutic use. In addition to its antidiabetic properties, it also produces a loss of appetite for alcoholic beverages. The alcoholic beverages may suddenly seem undesirable or even repugnant. Of 33 drinking diabetics on phenformin, 10 were affected in various degrees. With the anti-alcohol effect generally occurring very early in treatment, and on relatively low doses of between 75 and 150 mg daily, both the frequency and intensity of the effect vary with the dose. Case histories have indicated that a lack of desire for alcohol, anorexia, and alcohol indifference are all side effects found in phenformin therapy for diabetics. Since healthy persons can resist hypoglycemic effects of phenformin, it is suggested that, following further investigation, this drug may be suitable for case management of both diabetic and non-diabetic alcoholics.

797. Lish, P. M., Albert, J. R., Peters, E. L., and Allen, L. E.
 PHARMACOLOGY OF METHDILAZINE (TACARYL).
 Arch. Int. Pharmacodyn. (Gand), 129(1-2): 77-107 (27 ref.), 1960.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin.
 – in vivo – CNS – tranquilizers – *CAAAL-0 A-1418.

The pharmacological properties of methdilazine were investigated in in vivo and in vitro experiments on rats, dogs, mice, rabbits, and cats. The observed aspects of CNS action included: acute toxicity, conditional response, and anti-emetic, antipyretic, anti-Parkinsonian, gross reflex, and analgetic activities. The effects of methdilazine on sleeping time induced by sedatives, hypnotics, and anesthetics were investigated in mice. 6.18 mg/kg methdilazine (a dosage which had no observable hypnotic effect of its own) were administered ip, 30 min prior to ip injection of 23 ml 10% ethanol. Mean sleeping times of control and experimental groups were compared statistically. Alcohol sleeping time was significantly increased. In contrast to this, 28.4 mg/kg promethazine failed to significantly potentiate 23 ml 20% ethanol/kg. Certain components of methdilazine CNS actions, i.e., hypothermic activity and ability to intensify the effect of analgesics, hypnotics, and anesthetics, were quantitatively and qualitatively similar to those of chlorpromazine. The drug lacked those actions of chlorpromazine thought to be indicative of tranquilizing properties. The antihistamine action of methdilazine is perhaps the strongest and most selective of any phenothiazine. It also has strong anti-anaphylactic and antiserotonin actions.

798. List, P. H., and Reith, H.
 DER FALTENTINTLING, *COPRINUS ATRAMENTARIUS* BULL., UND SEINE DEM
 TETRAÄTHYLTHIURAMDISULFID ÄHNLICHE WIRKUNG. [The inky cap, *Coprinus*
atramentarius Bull., and its tetraethylthiuramdisulfide-like action].
 Arzneimittelforschung (Aulendorf), 10: 34-40 (14 ref.), 1960.
 G - ES - exp. comp. - DC (sensit.) - humans - acute admin. - in vivo - cardiovasc. - G.I. tract -
 respir. - senses - *CAAAL-0 A-1304.

The effects of *Coprinus atramentarius*, when taken with alcohol, are similar to those of calcium cyanamide and tetraethylthiuramdisulfide (TETD). Suspecting that the reason for this effect by *Coprinus* is the presence of TETD, the authors analyzed the mushroom, but found no TETD. Nor was the active ingredient found by separate administration of all the fractions resulting from the above analysis, together with alcohol, in a self-experiment. No fraction produced illness or the expected harmful effects. The 2 authors and another volunteer then each ate 250 g of freshly-picked mushrooms, and drank 100 ml red wine plus 40 ml dry gin; 2 of them ate the mushrooms raw, and 1 had them cooked. No immediate adverse effects ensued. The subject who ate cooked *Coprinus* drank a glass of beer 16 hr, and a glass of wine 24 hr, after the meal, whereupon sickness ensued lasting 1 night. The 2 others each drank 2 glasses of wine 24 hr after the meal, but neither felt ill. The authors conclude that either the effective agent cannot be absorbed from the mushroom in the raw state, or else it is only formed during cooking.

799. Lob, M.
 L'ACTION DU TRICHLOROÉTHYLÈNE SUR LE TAUX D'ALCOOL DANS LE SANG.
 [The influence of trichlorethylene on the blood alcohol level].
 Med. Lavoro (Milan), 51(10): 587-592 (13 ref.), 1960.
 F - ES - GS - IS - exp. cont. - DC (unchanged) - humans - mammals - acute admin. - in vivo
 - in vitro - blood lev. - analg., antipyret. - *CAAAL-9863-U1 A-0883.

The literature on trichlorethylene is reviewed. The author, in a self-experiment, plotted his own blood alcohol curve after consuming 10 cc of 80-proof whiskey. A few days later, he consumed the same amount of whiskey, 15 min after inhaling for 40 min a trichlorethylene concentration of 400 parts/million. No modification of the blood alcohol curve was found. A similar experiment was then conducted with rabbits. 4 rabbits received 1.3 cc absolute alcohol/kg in 10 cc water; 2 received 10 mg/kg trichlorethylene, diluted in a 2°/oo sol, followed 10 min later by 1.3 cc alcohol/kg; and 2 received alcohol and trichlorethylene simultaneously. The blood alcohol levels, determined after 10, 30, 90, and 150 min, were unaffected by the trichlorethylene.

800. Lockett, M. F., and Milner, G.
 COMBINING THE ANTIDEPRESSANT DRUGS.
 Brit. Med. J. (London), 1(5439): 921 (14 ref.), 1965.
 E – exp. – general – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS
 – antidepressants – *CAAAL-0 B-0360.

In a letter to the editor, the authors criticize a doctor who had previously stated in a letter to the same journal that it is “generally safe” to combine antidepressant drugs. It is reported that 2 patients died after normal therapeutic doses of amitriptyline, when followed by alcohol. In experiments with mice given amitriptyline (0.017 mg/10 g) and alcohol, amitriptyline potentiated the effect of alcohol on the righting reflex, and greatly increased the mortality caused by large doses of alcohol. Patients should be advised not to take alcohol when on antidepressants, pending further investigation.

801. Loitzl, E.
 ALKOHOL UND VERKEHRSSICHERHEIT. [Alcohol and traffic safety].
 Österreichische Ärztezeitung (Vienna), 18(11): 945-958 (55 ref.), 1963.
 G – SEC – review – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – psychol.
 perform. – CNS – senses – tranquilizers – *CAAAL-0 A-0884.

The literature on the topic is reviewed. Traffic statistics and laws of various countries are compared and commented on. The effects of alcohol and alcohol-drug combinations on the nervous system, the sensory organs, and on psychic reactions are discussed. Specific mention is made of alcohol in combination with chlorpromazine, which combination significantly strengthens neuromuscular reactions, and of alcohol with meprobamate, a combination reducing and impairing one's sense of judgment.

802. Loomis, H. P.
 THE TREATMENT OF ACUTE ALCOHOLISM BY LARGE DOSES OF DIGITALIS.
 J.A.M.A. (Chicago), 35(6): 337-338 (0 ref.), 1900.
 E – SEC – general – cross-tol. – drug-dep. humans – cardiovasc. – CNS – liver, kidney – skel., muscle,
 skin – cardiovasc. agents – *CAAAL-0 A-0885.

The author relates his experiences with the use of digitalis for the treatment of acute alcoholism. He states that the majority of investigators believe that the use of large doses of digitalis in acute alcoholism is not dangerous because of the cross-tolerance build-up. “It is interesting to note that a drug whose use even in moderately large doses is supposed to be fraught with some danger, can be given in one-half ounce doses without producing any of the ordinary symptoms of digitalis poisoning.” The usual dosage is 1/2 oz of tincture of digitalis every 4 hr for 3 doses. If the patient becomes quiet and the delirium disappears, the remedy is stopped. If not, another series of 3 doses 6 hr apart is given.

803. Loomis, H. P.
 THE TREATMENT OF ACUTE ALCOHOLISM BY LARGE DOSES OF DIGITALIS.
 Medical News (New York), 77(7): 239-241 (0 ref.), 1900.
 E – SEC – general – cross-tol. – drug-dep. humans – cardiovasc. – CNS – liver, kidney – skel., muscle,
 skin – cardiovasc. agents – *CAAAL-0 A-0886.

The author relates his experiences with the use of digitalis in the treatment of acute alcoholism. He states that the majority of investigators believe that the use of large doses of digitalis in acute alcoholism is not dangerous, because of the cross-tolerance build-up. “It is interesting to note that a drug whose use even in moderately large doses is supposed to be fraught with some danger, can be given in one-half ounce doses without producing any of the ordinary symptoms of digitalis poisoning.” The usual dosage is 1/2 oz of tincture of digitalis every 4 hr for 3 doses. If the patient

becomes quiet, and the delirium disappears, the remedy is stopped. If not, another series of 3 doses 6 hr apart is given.

804. Loomis, T. A.

EFFECTS OF ALCOHOL ON PERSONS USING TRANQUILLIZERS.

In: Havard, J.D.J., ed. *Alcohol and Road Traffic*. Proceedings of the Third International Conference on Alcohol and Road Traffic at London, September 3-7, 1962. London: British Medical Ass., pp. 1-4 (0 ref.), 1963.

E – exp. cont. – exp. comp. – presentation – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – mot. vehic. – acute admin. – in vivo – dose resp. – blood lev. – CNS – barbiturates – tranquilizers – *CAAAL-0 A-0887.

Male human subjects, who had performed simulated driving tasks at least 20 times under control conditions, were retested after po administration of tranquilizers alone or in combination with alcohol. In 1 test, 4 subjects were subjected to the following drug conditions at 1-week intervals: alcohol (100 ± 10 mg/100 ml) plus placebo, 25 or 50 mg secobarbital, 200 or 800 mg meprobamate, 25 mg chlorpromazine, and alcohol plus the individual tranquilizers. The 25 mg dose of secobarbital failed to affect performance, with or without alcohol, but the 50 mg dose significantly adversely altered performance when the blood alcohol level was 80-120 mg/100 ml. Both doses of meprobamate had effects similar to the 50 mg dose of secobarbital. The chlorpromazine failed to affect performance, with or without alcohol 2 hr after administration, but, after 6 hr, performance was significantly impaired. The author concludes that, if the dose is sufficiently great, the tranquilizer effect on performance will at least summate with the effects of alcohol.

805. Lundquist, F., Tygstrup, N., Winkler, K., and Jensen, K. B.

GLYCEROL METABOLISM IN THE HUMAN LIVER: INHIBITION BY ETHANOL.

Science (Washington), 150: 616-617 (9 ref.), 1965.

E – exp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – liver, kidney – metab. proc. – *CAAAL-11132-B1 B-0361.

Glycerol was administered to human subjects as a continuous infusion for a 50-min period, during the last half of which blood samples were taken. Ethanol was then added to the infusion during a further 50-min period, and blood samples again collected. There was, "a consistent reduction in the uptake of glycerol in the splanchnic area while ethanol was being administered to about 30% of the uptake in the control period." In 3 experiments in which ethanol was infused alone in the first period, and then infused with glycerol in the second period, no significant change in the rate of ethanol metabolism or output of acetate from the liver was noted. That a competition for alcohol dehydrogenase is responsible for the effect of ethanol on glycerol metabolism is ruled out, since glycerol is oxidized only at a negligible rate by liver alcohol dehydrogenase. The accumulation of glycerophosphate may be the cause of the inhibition, either by a direct inhibition of the kinase, or through a decreased ratio of adenosine triphosphate to adenosine diphosphate (which is known to influence the activity of this enzyme).

806. Lundsgaard, E.

GLYCEROL OXIDATION AND MUSCULAR EXERCISE.

Acta Physiol. Scand. (Stockholm), 12: 27-33 (3 ref.), 1946.

E – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – liver, kidney – metab. proc. – skel., muscle, skin – *CAAAL-4820-D1 A-0888.

2 male humans received glycerol (45 to 65 g), and the blood samples were analyzed. The decrease in blood glycerol concentration was slowed down during work. If ethanol (50 g po) was administered after glycerol, the glycerol oxydation was markedly decreased to about 1/3 of the normal rate. An

increase in the normal low glycerol concentration in the blood during muscular work, after inhibition of glycerol oxidation by the administration of ethanol, could not be demonstrated.

807. Lundt, P. V., and Jahn, E.

ALKOHOL BEI VERKEHRSSTRAFTATEN: ERGÄNZENDE STELLUNGNAHME ZU DEN BISHER VORGELEGTE GUTACHTEN DES BUNDESGESUNDHEITSAMTES ZUR FRAGE. [Alcohol in traffic offences: supplement to the evidence on the question thus far presented by the Federal Health Department].

Bad Godesberg: Kirschbaum, 39 pp. (147 ref.),

1967.

G – general – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – med.-leg. – mot. vehic. – humans – blood lev. – metab. proc. – analg., antipyret. – autocoids – barbiturates – elect., water-bal. agents – hormones, hormone antag. – sed., hypnot. – stimulants – *CAAAL-0 B-0362.

The question of influencing the metabolism of alcohol by various means, and the reliability of methods of alcohol determination in breath (alcotest, breathalyzer) are considered. The authors expound the problem of drugs and alcohol in automotive medicine, pointing out that their combined effect depends upon a number of organic functional variables, and, therefore, cannot be treated in general terms. It is stated that hitherto-publicized sobering agents are without practical value. It is further pointed out that barbiturates and psychopharmacological drugs, when in interaction with alcohol, are especially hazardous in automotive medicine, due to synergism. Insulin, used to accelerate alcohol decomposition and metabolism, is dangerous in drivers, because it may induce appreciable organic malfunctions by abruptly lowering the blood sugar level. Medical education and more stringent prescription regulations are necessary.

808. Luton

L'ALCOOLISME AU POINT DE VUE DE SES FORMES LARVÉES ET DE LA MÉDICATION STRYCHNIQUE. [Alcoholism from the point of view of its larval forms and of strychnine medication].

Bulletin Général de Thérapeutique Médicale, Chirurgicale, Obstétricale, et Pharmaceutique (Paris), 99: 241-250 (0 ref.),

1880.

F – general – DC (decrease) – humans – cardiovasc. – CNS – respir. – stimulants – *CAAAL-0 A-0889.

The author discusses the various forms and symptoms of alcoholism. The recommended dosage for delirium tremens is 3 cg strychnine sulphate several times/day, to a maximum of 15 or 20 cg over a 24-hr period. In referring to the various manifestations of alcoholism, it is admitted that the strychnine effect is not a true antidotal action if alcohol is not present in the system at the time of treatment, but, "Even with respect to these effects, we have, at least, an antagonistic action to exert; to oppose to sluggishness, stimulation; to the fall, the raising of temperature; to capillary hypemia, hyperemia; to regression, restoration; etc., all attributes which well belong to strychnine.... Here there are the marks of an incontestible physiological antagonism." One means of combatting alcoholism suggested by the author is the addition to certain beverages which are naturally bitter (e.g., absinthe, vermouth, etc.) of weak doses of nux vomica or strychnine under state supervision; these beverages would thus become more or less harmless, and would "merit preference" over those which are injurious.

809. Macaud, G.

LE TRAITEMENT DE L'ALCOOLISME PAR LE MAXITON. [Treatment of alcoholism with maxiton].

Gazette Médicale de France (Paris), 56: 31 (9 ref.),

1949.

F – general – DC (antidotal) – humans – CNS – amphetamines – *CAAAL-5900-N14 A-0890.

Maxiton (dextroamphetamine sulfate) is highly effective in acute alcohol poisoning (2 to 4 tablets), but less so in chronic alcoholism, although it alleviates the depression caused by alcohol. In 25% of the cases of chronic alcoholism, permanent weaning from the habit was possible (2 tablets mornings and evenings). It gives good results in delirium tremens (injection of 10 mg). There is no danger of addiction to maxiton after treatment.

810. MacCallum, W. A. G.

DRUG INTERACTION IN ALCOHOLISM TREATMENT.

Lancet (London), 1: 313 (0 ref.),

1969.

E – general – DC (sensit.) – drug-dep. humans – metab. proc. – antidepressants – tranquilizers – unclass. ther. agents – *CAAAL-13526 B-0573.

In a letter to the editor, the author points out how frequently citrated calcium carbimide (abstem) and disulfiram (antabuse) are used as 24-hr deterrents for the alcoholic. Frequently, the dosages are not sufficient for these drugs to be deterrents, and yet an increase of dosage may produce harmful side effects. However, if the patient is concomitantly given 25 mg amitriptyline (saroten) 3 times/day, then a sufficient deterrent reaction occurs without the side effects of either of the aforementioned drugs. The patient must also not be taking diazepam (valium), or possibly some other drugs, in order for amitriptyline to work properly. Presumably, the mode of action of amitriptyline is through a rapid build-up of the acetaldehyde blood level.

811. Macht, D. I., and Leach, H.

EFFECT OF METHYL AND ETHYL ALCOHOL MIXTURES ON BEHAVIOUR OF RATS IN A MAZE.

Proc. Soc. Exp. Biol. Med. (New York), 26: 330-331 (0 ref.),

1929.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – alcohols – *CAAAL-1688-J2 A-0891.

10 adult rats, trained to solve a maze problem, received, in the course of several hundred experiments, repeated 2 cc ip doses of: 2-4% ethanol sol, 2-4% methanol sol, or ethanol plus methanol (1:1). The effect on running time and the numbers of errors were noted. Ethanol was found to be definitely depressant, though not to a very great degree. Methanol had a definitely stimulating effect. The ethanol-methanol combination produced a very marked depression (running time and number of errors, respectively, were 122% and 120% of normal). A second series of experiments, involving a maze problem, also indicated that the toxicity of the 2 alcohols combined in equal proportions was more toxic than a double dose of either one alone.

812. Macht, D. I., and Davis, M. E.

SYNERGISTIC AND ANTAGONISTIC EFFECTS OF ALCOHOL, CAFFEINE AND NICOTINE MIXTURES.

Amer. J. Physiol. (Bethesda), 109: 67-68 (0 ref.),

1934.

E – abst. – exp. cont. – exp. comp. – DC (decrease) – mammals – other org. – acute admin. – in vivo – dose resp. – stimulants – *CAAAL-1414-D2 A-0892.

Phytopharmacological and zoopharmacological experiments were performed to determine synergism and antagonism. The individual toxicities and the toxicity of combinations of different pairs of sol of ethanol (0.5-1.0%), caffeine (1:1,000-1:2,000), and nicotine alkaloid (1:5,000-1:10,000) were studied with seedlings of *Lupinus albus*. Both the alcohol-caffeine and the alcohol-nicotine combinations were synergistic. Cats under ether anesthesia received sol of ethanol (0.52.0%), caffeine (1:2,000-1:5,000), and nicotine alkaloid (1:5,000-1:10,000). The alcohol-caffeine combination showed an antagonistic action, decreasing the toxicity of caffeine. The alcohol-nicotine combination was also antagonistic; the amount of nicotine required to produce death in such a combination was greater than that required when administered alone.

813. MacKenna, R. W.
 THE TOLERATION OF ARSENIC.
 Brit. Med. J. (London), 1: 85 (1 ref.), 1901.
 E – general – DC (add., infra-add., unspec. incr.) – humans – nerv. syst. – unclass. ther. agents –
 *CAAAL-0 A-0893.

The author presents statistics of 48 cases treated for skin problems with daily doses of up to 0.409 g of the double iodide of arsenic and mercury (Donovan's sol). With respect to arsenic poisoning from consumption of impure beer, it is doubted whether the small quantities of arsenic found in beer could produce peripheral neuritis; the alcoholic medium may possibly intensify the action of arsenic—there might be a chemical action between the menstruum and the salt, increasing the toxicity of the latter, or a combined toxic effect (since both alcohol and arsenic can produce peripheral neuritis), or the alcohol may interfere with the proper elimination of the drug by producing degenerative changes in various organs.

814. MacLeod, I.
 FATAL REACTION TO PHENELZINE.
 Brit. Med. J. (London), 1: 1554 (3 ref.), 1965.
 E – general – DC (add., infra-add., unspec. incr.) – post-mort. – humans – cardiovasc. – CNS – G.I.
 tract – respir. – barbiturates – enzymes – *CAAAL-0 B-0363.

The case history of a patient (age 44), hospitalized for anxiety and depression, and treated with phenelzine (15 mg, 3 times daily for 4 weeks before death), is presented. The fatal reaction which took place about 1 day prior to death resembled the "cheese reaction" type of adverse response to monoamine-oxidase inhibitors. The necropsy showed severe vascular congestion and oedema of the brain, with swelling of the cerebrum, and bilateral tentorial compression marks in the uncus region. The patient was a heavy drinker, and may well have ingested alcohol, in which case the alcohol would probably have been the precipitating factor. The evidence, however, is circumstantial, and the possibility of a spontaneous reaction from the drug alone cannot be ruled out.

815. MacLeod, L. D.
 THE CONTROLLED ADMINISTRATION OF ALCOHOL TO EXPERIMENTAL
 ANIMALS ("A MONTHLY BULLETIN" RESEARCH REPORT).
 Brit. J. Addict. (London), 45: 112-124 (4 ref.), 1948.
 E – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – amphetamines
 – *CAAAL-5043-D2 A-0894.

To determine the threshold at which some definite clinical signs of alcohol intoxication appear, rats were placed in a glass chamber with a constant alcohol-containing air current. Constant concentration of alcohol in chamber gas was 75 mg/l. "Intoxication threshold level" was established at a mean of 199 mg%, with a range of 118 to 259 mg%. On another occasion, rats were given 0.44 mg/kg dexedrine by stomach tube in 0.5 ml water, 1/2 hr before being placed in the inhalation chamber. No significant difference between the mean values of the intoxication threshold levels of blood alcohol for the group before, immediately after, or a week after dexedrine was noted. It is concluded that dexedrine was without influence on the reading of the blood alcohol levels at which a defined degree of impairment of coordination in the hind limbs of the rats took place.

816. MacLeod, L. D.
 "A MONTHLY BULLETIN" RESEARCH REPORT, 1948.
 Brit. J. Addict. (London), 46(1): 29-37 (5 ref.), 1949.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals –
 acute admin. – in vivo – mot. perform. – CNS – amphetamines – antispasmodics – autonomic agents

– coagulants – elect., water-bal. agents – gastrointest. agents – miscellaneous – musculoskel. agents
 – nutritive agents – sed., hypnot. – stimulants – unclass. ther. agents – *CAAAL-5120-D2

A-0895.

Autoselection experiments, tests for intoxication, and biochemical (intermediary metabolism) studies with the Warburg apparatus were conducted. In the intoxication tests, groups of 30 rats (in sub-groups of 10—1 sub-group serving as control) were subjected to chronaximetric and grid tests for intoxication. Alcohol was administered by stomach tube, in a dose found to produce signs of moderate intoxication. The following substances were given by stomach tube or injection 15 min prior to the alcohol, in doses calculated on a body wt basis from the maximal therapeutic dose: atropine, carbaminoyl choline, eserine, pilocarpine, prostigmine, adenylic acid, nicotinamide, thiamine, amphetamine, apomorphine, caffeine, ephedrine, alanine, glucose, glutamic acid, sodium acetate, sodium citrate, sodium pyruvate, and sodium ethyl phosphate. None of the substances tested had a marked effect on the degree of intoxication, and only prostigmine appeared to show a definite tendency to increase it.

817. MacLeod, L. D.

“MONTHLY BULLETIN” RESEARCH REPORT, 1949.

Brit. J. Addict. (London), 47(1): 48-61 (16 ref.),

1950.

E – SEC – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – miscellaneous
 – *CAAAL-5608-B2

A-0896.

Coccarboxylase activity of the brain and pyruvate metabolism in alcohol intoxication, acetaldehyde intoxication and the effects of tetraethylthiuramdisulphide, and maze studies using rats were investigated. Maze work, to be published elsewhere, was conducted with rats after simultaneous administration of acetaldehyde and alcohol. It appeared from the worsened state of the animals, and particularly from the time relationships involved, that something very like synergistic action took place. This indicates that the action of acetaldehyde may be different, according to whether or not alcohol is present. Later observations, however, raised the possibility that delayed effects of anoxia may have been observed under the experimental conditions used.

818. MacLeod, L. D.

MONTHLY BULLETIN RESEARCH REPORT, 1950.

Brit. J. Addict. (London), 49(1-2): 60-69 (9 ref.),

1952.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – cardiovasc.
 – CNS – metab. proc. – autonomic agents – barbiturates – musculoskel. agents – sed., hypnot. –
 *CAAAL-6245-B2

A-0897.

In 1 experiment on rats, the effect of alcohol upon acetylation of p-aminobenzoic acid was studied. In another experiment, rats weighing 160 g were given 3 ml 30% alcohol by stomach tube, and the degree of intoxication assessed by maze running and grid iron tests. 0.1 mg carbachol was administered alone or 1/2 hr prior to the alcohol. It was found that, whereas the alcohol produced very slight impairment, and the carbachol produced no observable impairment, the carbachol-alcohol combination resulted in profound and prolonged intoxication. Prostigmine (neostigmine) also had this effect in combination with alcohol, as did di-isopropyl fluorophosphate to a lesser degree. Tetraethyl pyrophosphate and eserine (physostigmine) were found to be too toxic for this type of experiment.

819. MacLeod, L. D.

“A MONTHLY BULLETIN” RESEARCH REPORT.

Brit. J. Addict. (London), 50(2): 89-135 (109 ref.),

1953.

E – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – CNS – liver, kidney – metab. proc. – gastrointest. agents – *CAAAL-6714-B10

A-0898.

The author reports on research conducted on the alcohol intoxication process and on the choline dehydrogenase enzyme of the rat liver, then gives a general review of work in the field by other researchers. Tests on maze-trained rats were conducted with various substances, including succinate, fumarate, malonate, and maleate. At non-toxic levels, it was found that succinate and malonate were without effect on ethanol intoxication, whereas fumarate to a slight degree, and maleate to a more marked extent, appeared to exert a slight protective action against intoxication. Rats were then pretreated with 75 mg sodium maleate/day by stomach tube for 6 days, followed by a dose of 6 ml 30% ethanol. The results showed that the rats receiving maleate were only slightly intoxicated (i.e., incoordination was detectable by the vertical grid test but was not usually apparent when the animal was walking under normal conditions). The control rats which received only ethanol, were deeply intoxicated, normally to the extent of helplessness or complete unconsciousness.

820. MacLeod, L. D.

MONTHLY BULLETIN RESEARCH REPORT.

Brit. J. Addict. (London), 53(2): 139-143 (0 ref.),

1957.

E – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – acid-base, blood pH, elect. – CNS – anti-infectants – autonomic agents – sed., hypnot. – *CAAAL-7757-B2

A-0899.

The author reports on his studies concerning permeability and related phenomena in the rat brain. Albino rats were given trypan blue iv to stain the “blood-brain” barrier, after being intoxicated by alcohol alone or in combination with carbachol. The animals were then killed and the brains examined. There was no indication of impairment in vivo caused by either alcohol or alcohol-carbachol. The effect of carbachol in augmenting alcohol intoxication is striking, but the author concludes that, if an explanation for this is sought in terms of the permeability changes occurring at the cell surface or other membranous structures, present studies suggest that, while such changes may add to the disorganization of function resulting from the action of alcohol within the cells, the immediate sites of action of alcohol and carbachol are probably different.

821. MacMahon, H. E., and Weiss, S.

CARBON TETRACHLORIDE POISONING WITH MACROSCOPIC FAT IN THE PULMONARY ARTERY.

Amer. J. Path. (New York), 5: 623-630 (11 ref.),

1929.

E – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – drug-dep. humans – cardiovasc. – liver, kidney – anti-infectants – *CAAAL-0

A-1305.

An alcoholic labourer, following a 3-day binge, had taken carbon tetrachloride in milk, and exhibited symptoms of continuous abdominal pain, hiccoughs, nausea, and vomiting; thereafter, he again ingested carbon tetrachloride, became semicomatose and was admitted to hospital. The patient was intensely jaundiced, vomited a foul-smelling dark fluid material (probably old blood), had severe halitosis, and his mouth and tongue were coated with a brownish material. Life signs continued to diminish, and death followed 48 hr after admission. Autopsy showed extensive degeneration of central and mid-zones of liver lobules, with fat globules in this tissue and also filling the hepatic veins; fat was found in the kidney glomeruli and collecting ducts and in the brain capillaries; the pulmonary blood was 64% pure fat by vol with 25% pure fat in the inferior vena cava blood. The patient's liver was that of early alcoholic cirrhosis, large and fatty, and the effect of the liver degeneration and necrosis by the carbon tetrachloride, and the subsequent release of pure fat into the pulmonary circulation and various organs, produced the pathology at autopsy. “It appears from these observations that the sensitiveness of alcoholic patients to carbon tetrachloride is increased not only because of increased absorption of the drug, as well as the synergistic action of alcohol and carbon tetrachloride together in producing liver damage, but also because of the possible presence of preexisting liver damage.”

822. Madan, B. R., Sharma, J. D., and Vyas, D. S.
SOME NEUROPHARMACOLOGICAL ACTIONS OF LIBRIUM.
 Annals of Biochemistry and Experimental Medicine (Calcutta), 22(9): 221-224 (14 ref.), 1962.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS –
 tranquilizers – *CAAAL-0 A-0900.

Librium (5 mg/kg ip) was administered to 45 rats, while a control group received distilled water. 15 min later, 2 g/kg ethanol ip was given to both groups. Hypnosis was not induced with ethanol alone, but, with the ethanol-librium combination, sleep was produced for 46.0 ± 4.3 min. The interaction of librium with pentobarbital, pentylenetrazol, picrotoxin, and strychnine was also investigated. Librium should be contraindicated in cases of alcoholic intoxication, and patients on treatment should be warned against drinking alcohol.

823. Madan, B. R., and Gupta, R. S.
EFFECT OF ETHANOL IN EXPERIMENTAL AURICULAR AND VENTRICULAR ARRHYTHMIAS.
 Jap. J. Pharmacol. (Kyoto), 17: 683-684 (7 ref.), 1967.
 E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo –
 dose resp. – cardiovasc. – skel., muscle, skin – barbiturates – *CAAAL-0 B-0958.

Atrial fibrillation was produced in 6 pentobarbitalized (30 mg/kg ip) dogs by topical application of 5% acetylcholine on the sinus node. Ventricular arrhythmias were produced by another method in 10 dogs. The anterior descending branch of the left coronary artery was ligated in 2 stages under pentobarbital anesthesia. Ethanol (500 mg/kg diluted to 20 ml) was given ip over 10 min, and repeated every 1/2 hr if necessary, up to 3 injections. In the acetylcholine group, 500 mg/kg ethanol caused a 45.2% reduction in duration of arrhythmia. In ectopic ventricular tachycardia, ethanol reduced both the total heart rate and ectopic ventricular beats in all experiments. The blood concentration of ethanol which coincided with maximal antiarrhythmic effect was 125 mg%. Thus, ethanol at blood concentrations which are attainable in man exerts a beneficial effect in auricular and ventricular arrhythmias. The mechanism is not known, as alcohol does not share the electro-physiological properties of other antiarrhythmics.

824. Madsen, J., and Larsen, J. A.
TOLBUTAMIDS INDFLYDELSE PÅ ALKOHOLMSAETNINGEN. [The effect of tolbutamide on alcohol metabolism].
 Nord. Med. (Stockholm), 66: 1540-1542 (19 ref.), 1961.
 S – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood
 lev. – other drug lev. – liver, kidney – metab. proc. – hormones, hormone antag. – *CAAAL-9888-A2
 A-1306.

The effect of tolbutamide and carbutamide on ethanol metabolism was studied in anesthetized cats. Sufficient alcohol to maintain a constant plasma concentration of 50-150 mg/l was administered iv to 8 cats, and 1 hr was allowed for diffusion equilibrium to occur. Blood samples were taken every 1/2 hr for 2 hr before and after the iv injection of 100 mg/kg body wt of tolbutamide. In similar experiments, 3 cats were administered carbutamide, and 4 others received physiological water. It was found that ethanol metabolism after tolbutamide administration was reduced by a mean of 35% (range 17-50%). After carbutamide administration, 1 cat showed a 24% reduction in ethanol metabolism, whereas the other 2 cats and those which received physiological water showed no change. When 100 mg/kg of S^{35} -tolbutamide was given to cats, the plasma concentration during the first 2 hr was 30-35 mg/100 ml. It is concluded that ethanol metabolism is inhibited by tolbutamide and, to a lesser extent, carbutamide.

825. Magnussen, M. P.
 THE INFLUENCE OF ETHANOL ON THE ABSORPTION OF DRUGS FROM THE RAT SMALL INTESTINE.
 Acta Pharmacol. (Copenhagen), 25(Suppl. 4): 40 (2 ref.), 1967.
 E – exp. cont. – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – G.I. tract – barbiturates – neoplast. agents – sed., hypnot. – *CAAAL-0 B-0364.

The influence of ethanol on the absorption of barbitone, phenobarbitone, pentobarbitone, promethazine, and sulphaguanidine from the rat small intestine was investigated. The small intestine was perfused with purely aqueous or alcoholic (0.5, 1.0, or 2.0%) drug sol. In no case did alcohol have any effect on the absorption. In another series of experiments, a steady state of blood alcohol concentration of 1 or 1.5 mg/ml was shown to have no effect on the absorption of barbitone and promethazine.

826. Magnussen, M. P., and Frey, H. -H.
 EINFLUSS VON ALKOHOL AUF DIE RESORPTION ZENTRAL DEPRESSIVER PHARMAKA AUS DEM MAGEN. [Influence of alcohol on the absorption of centrally acting depressants from the stomach].
 Naunyn Schmiedeberg. Arch. Pharm. Exp. Path. (Berlin), 257(1): 39 (0 ref.), 1967.
 G – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – blood lev. – absorp., distrib., stor. – CNS – G.I. tract – barbiturates – gastrointest. agents – sed., hypnot. – *CAAAL-0 B-0365.

The effect of alcohol on the absorption-time of pentobarbital, phenobarbital, and promethazine from the stomach was investigated in rats. In the first experiment, pentobarbital and phenobarbital were administered into the stomach in sol containing 0-20% alcohol (w/v), and, within 1 hr, the absorbed drug percentage was determined. For 0, 1, 2.5, 5, 10, 15, and 20% alcohol sol, the absorbed percentages of pentobarbital were 23.7, 24.4, 31.7, 30.1, 28.0, 25.3, and 21.4% respectively, and, for 0, 1, 2.5, 5, and 10% alcohol sol, the absorbed phenobarbital percentages were 17.1, 20.7, 20.1, 24.8, and 22.3%, respectively. It was shown that alcohol percentages of 1-10% stimulated drug absorption, and higher percentages decreased it. In a second experiment, the blood alcohol level was maintained at 1.5°/oo. Compared with untreated controls, the absorption of pentobarbital was increased from 20.8 to 26%, and phenobarbital absorption increased from 13.8 to 19.9%. Promethazine was absorbed neither in the presence nor in the absence of alcohol.

827. Magnussen, M. P.
 THE EFFECT OF ETHANOL ON THE GASTROINTESTINAL ABSORPTION OF DRUGS IN THE RAT.
 Acta Pharmacol. (Copenhagen), 26(2): 130-144 (26 ref.), 1968.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – in vitro – blood lev. – absorp., distrib., stor. – acid-base, blood pH, elect. – cardiovasc. – G.I. tract – barbiturates – gastrointest. agents – sed. hypnot. – *CAAAL-0 B-0366.

In vivo absorption from the stomach and small intestine of female rats was studied with drug sol containing 200 µg/ml of the drug concerned. In 1 experiment on the stomach, ethanol (1-20% (w/v) concentration) was present in sol of phenobarbital, pentobarbital, and promethazine. In another experiment on the stomach, a priming dose of 1.05 g/kg ethanol iv was given, and a blood ethanol concentration of 1.5 mg/ml maintained during absorption of the above drugs. In an experiment on the small intestine, ethanol was present in concentrations of 0.5, 1.0, or 2.0% (w/v), in sol of barbital, phenobarbital, pentobarbital, sulphaguanidine, and promethazine which were perfused into the intestine, and, in another experiment, after a priming dose of 1.05 g/kg ethanol iv, a concentration of 1-1.5

mg/ml ethanol was maintained during perfusion of the intestine by barbitol and promethazine. In vitro experiments were conducted on isolated segments of jejunum perfused with sol containing 1 mM pentobarbital or 400 μ g/ml sulphaguanidine; the effect of ethanol was determined when present on the serosal side (1.5 mg/ml) or on the mucosal side (in the drug sol: 1.0 or 2.0% (w/v)). It was found that ethanol is capable of significantly enhancing absorption from the stomach of those drugs which are well absorbed without ethanol. Ethanol failed to influence drug absorption from the small intestine or isolated jejunum segments.

828. Mallach, H. J.

EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE WIRKUNG EINES ALKOHOL-ERNÜCHTERUNGSMITTELS. [Experimental investigations on the effect of a sobering-up agent].

Arzneimittelforschung (Aulendorf), 9(6): 389-390 (7 ref.), 1959.
G – ES – exp. – DC (unchanged) – humans – acute admin. – in vivo – in vitro – blood lev. – absorp., distrib., stor. – CNS – senses – *CAAAL-9044-A1 A-0901.

Promill-Ex, a commercial product containing lecithin, essential fatty acids, catalytic enzyme systems from yeast, coffee extract, vitaminized albumins, and vegetable extracts, was tested in 12 healthy humans as a sobering-up drug. It was tested on the nystagmus and reaction time of 12 humans who received 1 capsule of the drug on 1 day for every 20 g of absolute alcohol, and on the second day for every 7 g of absolute alcohol. It was concluded that the drug had no effect on absorption or elimination of alcohol, and did not improve the reaction time towards optic or acoustic stimuli following the application of alcohol. In vitro tests did not indicate enzymatic action on alcohol metabolism.

829. Mallach, H. J., and Röseler, P.

BEOBACHTUNGEN UND UNTERSUCHUNGEN ÜBER DIE GEMEINSAME WIRKUNG VON ALKOHOL UND KOHLENMONOXYD. [Observations and studies on the combined action of alcohol and carbon monoxide].

Arzneimittelforschung (Aulendorf), 11(11): 1004-1008 (29 ref.), 1961.
G – ES – exp. – stat. surv. – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – blood lev. – blood comp., sites, lymph – indust. intox. – *CAAAL-10699-D1 A-0902.

Among 1610 forensic autopsies performed at the University of Berlin from 1956 to 1960 were 72 (4.5%) carbon monoxide poisonings. In 13 cases of combined carbon monoxide (CO) and alcohol poisoning, the blood alcohol concentration was 3.9-4.2°/oo. Observations made in humans who had died from combined effects of both compounds showed a synergism of CO and alcohol. Although the lethal alcohol dose for mice (8 g/kg) is twice the lethal dose of humans (3.0-3.5 g/kg), experiments with mice also showed synergism. Synergism between CO and alcohol may lead to driving impairment, but no reliable evidence exists at present.

830. Mallach, H. J., and Etzler, K.

TIEREXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE GEMEINSAME WIRKUNG VON DIMETHYLSULFOXYD UND ÄTHYLALKOHOL. [Animal experiments on the joint action of dimethylsulfoxide and ethyl alcohol].

Arzneimittelforschung (Aulendorf), 15(11): 1305-1308 (14 ref.), 1965.
G – ES – exp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – *CAAAL-0 B-0367.

The mean lethal dose of dimethylsulfoxide (DMSO) was determined in mice by administering the substance po in a concentrated form (100%). The values determined for the 24-hr limit were 24.2 g/kg, and for the 72-hr limit were 23.1 g/kg. These values are valid for an ambient temperature of

24°C. With simultaneous alcohol-DMSO administration, the mortality quota of the alcohol LD₅₀ was reduced with small DMSO doses (2.4 g/kg). On the other hand, small amounts of alcohol had no influence on the mechanism of action of the DMSO LD₅₀. No serious objections are raised against testing the joint action of DMSO (50 mg/kg cutaneously) and alcohol (0.75 g/kg po) in humans.

831. Mallach, H. J.

GEMEINSAME WIRKUNG VON AETHYLALKOHOL UND DOLICUR. [Interaction of ethyl alcohol and dolicur].

In: Laudahn, G., et al., eds. *Dimethylsulfoxyd—DMSO: Symposium am 2. Juli 1965 in Berlin*. Wissenschaftliche Abteilungen der Schering AG. [Dimethylsulfoxide—DMSO: symposium on July 2, 1965 in Berlin. Scientific departments of Schering AG]. Berlin: Schering AG., pp. 27-31 + "Discussion", pp. 32-42 (0 ref.), 1965.

G – exp. cont. – presentation – DC (decrease) – humans – mammals – acute admin. – in vivo – dose resp. – *CAAAL-0 B-0368.

The interaction of dimethylsulfoxide (DMSO) (24.0 g/kg) and alcohol (8.4 g/kg, LD₅₀ = 1.000) was examined in mice. It was found that the lethal quotient (alcohol 1.000/DMSO 0.100) receded to 30%. The average survival at a combination of 1.000/1.000 was 143 min. At decreased doses of DMSO, survival time increased, and it is concluded that lower DMSO doses decrease the effect of alcohol in animals. In another experiment, 5 young men drank 0.75 g/kg alcohol in 10 min, and then 50 mg/kg DMSO was immediately applied cutaneously. The only difference with controls (who received alcohol only) was in the hourly decrease value (β_{60}), which was 0.170°/oo for alcohol-DMSO and 0.128°/oo for alcohol only. With some subjects receiving alcohol-DMSO, no alcohol odour on the breath could be detected, and it is speculated whether or not breathalyzer tests could be affected.

832. Mallach, H. J.

METHODISCHE UNTERSUCHUNGEN ZUR QUANTITATIVEN ERFASSUNG DER GEMEINSAMEN WIRKUNG VON ALKOHOL UND ARZNEIMITTELN. [Methodological investigation on the quantitative analysis of the interaction of alcohol and drugs].

Blutalkohol (Hamburg), 4(4): 165-169 (17 ref.), 1967.

G – general – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – anticonvulsants – sed., hypnot. – *CAAAL-0 B-0369.

The development of variance analysis during the years 1963-1967 is reviewed. The method of testing different alcohol-drug combinations is described and illustrated. It is stated that this method is well suited to establish single and combined effects of alcohol and drugs, and is useful in evaluating driving ability. No original experiments are reported.

833. Mallach, H. J.

INTERACTION OF DMSO AND ALCOHOL.

Ann. N.Y. Acad. Sci. (New York), 141: 457-462 (14 ref.), 1967.

E – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – dose resp. – blood lev. – other drug lev. – metab. proc. – skel., muscle, skin – *CAAAL-0 B-0370.

1900 white mice received alcohol and dimethylsulfoxide (DMSO) po combined in their mean lethal doses and fractions thereof, in such a way that the dose of one was kept constant while the other was varied. DMSO given simultaneously in small doses reduced mortality from alcohol from 50% to 37%, but alcohol failed to affect mortality from high DMSO doses. When alcohol was given 1 hr after DMSO, mortality was doubled, and, when DMSO was given 1 hr after alcohol, mortality was increased fourfold. 46 human subjects received 0.75 g alcohol/kg, and 50 mg DMSO/kg was applied to the skin of the back simultaneously with the alcohol ingestion, or 1 hr prior to alcohol. It was found

that the mean decline/hr of the blood alcohol level was increased 28% by the simultaneous combination, and was increased 17% when DMSO was given 1 hr before alcohol. It is concluded that the critical factor is the time relationship of application. DMSO does not accelerate alcohol absorption, but does increase the rate of decline of the blood alcohol level.

834. Mallach, H. J.

ALKOHOL UND ARZNEIMITTEL ALS VERKEHRSMEDIZINISCHES PROBLEM.

[Alcohol and drugs as a medical problem in traffic].

Materia Medica Nordmark (Hamburg), 20(8): 430-436 (31 ref.),

1968.

G – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – mammals – metab. proc. – tranquilizers – *CAAAL-0 B-0371.

The paper gives a statistical analysis and evaluation of the problems of interaction between alcohol and drugs in relation to automotive medicine. To answer the question whether such interactions are detrimental to traffic safety, the author advocates the development of supersensitive analytical methods for examining the pharmacokinetics, and of statistical models to study interactions between 2 or more substances. References are made to a test method for determining the individual factors influencing the interaction mechanism between alcohol and DMSO, or between alcohol and psychopharmacological drugs in white mice. In conclusion, the author remarks that numerous questions remain as yet unanswered, e.g., whether drugs induce genuine potentiation of the alcohol effect.

835. Malone, M. H., Gibson, R. D., and Miya, T. S.

A PHARMACOLOGIC STUDY OF THE EFFECTS OF VARIOUS PHARMACEUTICAL VEHICLES ON THE ACTION OF ORALLY ADMINISTERED PHENOBARBITAL.

J. Amer. Pharm. Ass. (Chicago), 49: 529-534 (10 ref.),

1960.

E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – species or sex diff. – respir. – barbiturates – *CAAAL-0 A-0903.

The pharmacodynamics of phenobarbital elixir U.S.P. XIV (containing phenobarbital, ethanol, and glycerol) was studied in rats and mice. Calculations utilizing log transformation data were used to accurately summarize the results obtained. It was impossible to determine the relative merit of the intact elixir, because the narcotic effect of phenobarbital was qualitatively modified by the various actions of both ethanol and glycerol. Various combinations of ethanol and glycerol were selected in which 182.7 mg/kg of phenobarbital could be dosed at 5 cc/kg; with only four rats dosed per test treatment, only qualitative conclusions could be drawn which corroborate the findings of Hazelton, Lloyd W., and Hellerman, Rebecca C. (J. Amer. Pharm. Ass. (Washington), 35(6): 161-168, 1946).

836. Manno, J. E., Kiplinger, G. F., Bennett, I., and Forney, R. B.

HUMAN MOTOR AND MENTAL PERFORMANCE UNDER THE INFLUENCE OF ALCOHOL AND/OR MARIHUANA.

Toxic. Appl. Pharmacol. (New York), 17(1): 306 (0 ref.),

1970.

E – abst. – exp. comp. – DC (unspec.) – humans – acute admin. – in vivo – dose resp. – other drug lev. – mot. perform. – psychol. perform. – CNS – hallucinogens – *CAAAL-0 B-0959.

Experiments were conducted to establish a dose-response relationship for human mental and motor performance after smoking marihuana, and to test the effect of marihuana combined with alcohol. 12 male volunteers were each administered on 6 different occasions a marihuana cigarette calibrated to deliver in the smoke approximately 0, 2.5, or 5 mg delta-9-tetrahydrocannabinol. In addition, a flavoured beverage, with or without alcohol (sufficient alcohol to produce a blood alcohol concentration of 0.05%), was consumed over a 30-min period prior to the marihuana. Combined drug administrations were randomized and double-blind. Pulse rates and alveolar breath alcohol levels were determined periodically throughout the experimental period, and, 30 min after the start of the

marihuana smoking, performance testing was begun. Marihuana components in the cigarettes and cigarette remains were analyzed by gas chromatography. The experimental results are not stated.

837. Manno, J. E., Kiplinger, G. F., Scholz, N., and Forney, R. B.
 THE INFLUENCE OF ALCOHOL AND MARIHUANA ON MOTOR AND MENTAL PERFORMANCE.
 Clin. Pharmacol. Ther. (St. Louis), 12(2): 202-211 (11 ref.), 1971.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – dose resp. – blood lev. – mot. perform. – psychol. perform. – cardiovasc. – CNS – senses – hallucinogens – *CAAAL-0 B-0960.

The effects of marihuana, alone or in combination with alcohol, on mental and motor performance, were studied in a double-blind randomized experiment on 12 healthy men. All subjects received 6 treatments within 1-week intervals. Each treatment consisted of a 30-min drinking period, followed by a test cigarette, after which 4 pursuit meter tests, 9 delayed auditory feedback (DAF) tests, pulse rate, conjunctival injection, and subjective effects (Cornell Medical Index) were recorded. The cigarettes were calibrated to deliver 0, 2.5, or 5 mg delta-9-tetrahydrocannabinol (THC), alone or in combination with a plain fruit-flavoured beverage, or the same beverage containing 15 ml alcohol/50 lb body wt. The combination of marihuana and alcohol, as compared to marihuana alone, generally impaired pursuit meter and DAF performance, but, in most cases, not significantly so. The decrement produced by the combination was significantly greater than alcohol or marihuana alone in the first pursuit meter pattern, and in reverse verbal output on the DAF. Alcohol enhanced both the intensity and duration of marihuana effects on eye and pulse changes, and additively increased subjective effects. It is concluded that alcohol and marihuana exert additive effects on performance.

838. Mardones, J., Cembrano, J., and Muñoz, E.
 EFFECT OF VOLUNTARY INTAKE OF ETHANOL ON THE SURVIVAL TIME OF RATS SUBMITTED TO CHRONIC INTOXICATION WITH CARBON TETRACHLORIDE.
 Acta Physiol. Lat. Amer. (Buenos Aires), 11: 268-269 (0 ref.), 1961.
 E – abst. – exp. cont. – DC (decrease) – DC (unchanged) – mammals – chronic admin. – in vivo – anti-infectants – *CAAAL-0 A-0904.

4 groups of 6 rats each were submitted to chronic intoxication with carbon tetrachloride (1 min exposure to carbon tetrachloride vapour-saturated atmosphere/day). 2 groups belonged to the “drinker” strain and 2 others to the “non-drinker” strain. No influence of alcohol intake on the survival time of the rats of the “non-drinker” strain was observed, but the survival time of rats of the “drinker” strain which received alcohol was significantly higher than both the two “non-drinker” groups and the “drinker” one which did not receive alcohol.

839. Mardones, J.
 THE ALCOHOLS.
 In: Root, Walter S., et al., eds. *Physiological Pharmacology. I.* New York: Academic Press, pp. 99-183 (599 ref.), 1963.
 E – review – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – blood lev. – absorp., distrib., stor. – CNS – metab. proc. – respir. – amphetamines – analg., antipyret. – autonomic agents – barbiturates – miscellaneous – musculoskel. agents – stimulants – tranquilizers – *CAAAL-0 A-0905.

The alcohol literature is reviewed. Topics covered include: the metabolism of alcohol, the effect of alcohols on the central nervous system, the effect of alcohols outside the central nervous system, synergism and antagonism between alcohols and other drugs (i.e., barbiturates, morphine, phenothia-

zine derivatives, epinephrine, cortisone, pentetrazole, nikethamide, picrotoxin, amphetamine, acetaldehyde, strychnine, and cocaine), appetency for ethanol, effect of prolonged use of alcohols, and the toxicology of alcohols.

840. Mardones, J., and Solodkowska, W.

**EINFLUSS CHRONISCHER VERGIFTUNG MIT TETRACHLORKOHLENSTOFF
AETHANOLSTOFFWECHSEL IN LEBER UND FETTGEWEBE BEI DER RATTE.**

[Influence of chronic poisoning with carbon tetrachloride on ethanol metabolism in liver and fatty tissue of the rat].

In: Glass, Theo, et al., eds. *Alkohol und Alkoholismus: 27 Internationaler Kongress*. [Alcohol and alcoholism: 27th International Congress]. Hamm, Westf., West Germany: Deutsche Hauptstelle gegen die Suchtgefahren, pp. 130-131 (O ref.), 1965.

G – exp. cont. – presentation – DC (unchanged) – mammals – chronic admin. – in vivo – in vitro – species or sex diff. – liver, kidney – metab. proc. – anti-infectants – *CAAAL-0 B-0372.

Previous experiments showed that carbon tetrachloride (CCl₄)-poisoned rats eliminate alcohol from the blood faster than controls, although there is no difference in the rate of oxidation. To investigate whether this difference is due to an increased fat synthesis in the liver or fatty tissues, the activity of alcohol was studied in the eliminated carbon dioxide, in the fat of the liver, in in vitro fatty tissue slices (after 2 hr of incubation), and in 80 male and female albino rats from “drinker” and “non-drinker” strains. The experimental group received 10% v/v sol of 95% alcohol in water, and, for 12 weeks, were exposed once a day for 1 min to CCl₄ vapour. The controls received only the alcohol. After 12 weeks, the rats receiving CCl₄ were killed, and liver slices were investigated. The chronic CCl₄ administration seemed to have had no effect on the capacity of the liver slices to oxidize alcohol. Only the females of the “non-drinker” strain had a higher oxidation rate than the males of the same strain.

841. Mareček, P., and Bakalář, E.

POKUS O OVLIVNĚNÍ PRŮBĚHU INTOXIKACE LSD ETYLALKOHOLEM. [An attempt to influence the course of LSD intoxication with ethyl alcohol].

Activ. Nerv. Sup. (Prague), 9(4): 379-380 (O ref.),

1967.

C – exp. cont. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – psychol. perform. – cardiovasc. – metab. proc. – senses – hallucinogens – *CAAAL-0 B-0373.

To determine the effect of ethanol on LSD intoxication, 6 human subjects received 1 g/kg ethanol po in the form of a 38% distillate, and 100 µg LSD under the following conditions: LSD plus ethanol 1 hr later, LSD plus placebo, and ethanol alone. Physiological and psychological parameters were observed. With respect to the ethanol-LSD combination, it was found that a significant difference occurred in the number of symptoms indicating perceptual differences (colour, sound, time, etc.). Physiological measurements showed that the pulse rate is substantially accelerated, reaching maximum rates in the third, fifth, and sixth hr. 1 1/2 hr after the beginning of the experiment, until the end, the number of symptoms of vegetative differences appeared to be higher than placebo controls. It is concluded that ethanol is not antagonistic to LSD intoxication, but does modify the course of the intoxication.

842. Markham, T. N.

RENAL FAILURE DUE TO CARBON TETRACHLORIDE.

J. Occup. Med. (New York), 9: 16-17 (5 ref.),

1967.

E – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – humans – other drug lev. – acid-base, blood pH, elect. – blood comp., sites, lymph – cardiovasc. – G.I. tract – liver, kidney – respir. – anti-infectants – *CAAAL-12061-D1 B-0374.

A 29 yr-old man was exposed to carbon tetrachloride (CCl₄) vapours for 1/2 hr while cleaning tiles. During the period of exposure, he had drunk 2 cocktails, and his history revealed an alcoholic intake of 1/2-1 pint of whiskey/day for 6 yr. 8 days after exposure, he was admitted to hospital, and he died on the 14th day of hospitalization. A postmortem examination revealed pulmonary congestion of the left lung, marked fibrinopurulent exudate on all peritoneal surfaces, centrilobular necrosis of the liver with collapse of the reticulum and slight fatty infiltrates, and, in the kidney, a flattening of the tubular epithelium, some hyalinized glomeruli, and acute passive congestion. The author comments on the possible mechanism of potentiation of CCl₄ toxicity by alcohol.

843. Marquis, D. G., Kelly, E. L., Miller, J. G., Gerard, R. W., and Rapoport, A.
 EXPERIMENTAL STUDIES OF BEHAVIORAL EFFECTS OF MEPROBAMATE ON
 NORMAL SUBJECTS.
 Ann. N.Y. Acad. Sci. (New York), 67: 701-711 (0 ref.), 1957.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mot. vehic. – humans –
 acute admin. – in vivo – mot. perform. – psychol. perform. – species or sex diff. – glands – senses
 – nutritive agents – tranquilizers – *CAAAL-7874-J1 A-0906.

On 5 successive days, 50 human subjects were treated with 1 of the following: placebo, meprobamate (800 mg), dextroamphetamine sulfate (15 mg), meprobamate plus alcohol (2 oz 80-proof whiskey), and placebo plus alcohol. Effects of the drugs on accuracy, speed, judgment, reaction time, steadiness, and vision were studied, using a simulated driving task. The results showed that meprobamate alone, even in double the usual dosage, produces no behavioral toxicity. Whereas alcohol definitely impairs performance on some tests, the combination with meprobamate does not significantly add to the alcohol impairment. The data “give no grounds for preventing persons under the usual dosages of meprobamate from driving automobiles, or even from driving under meprobamate after drinking alcohol in amounts that would not ordinarily affect driving ability.”

844. Marseille
 EMPOISONNEMENT PAR L'ALCOOL.—EMPLOI DE L'ACÉTATE D'AMMONIAQUE;
 GUÉRISON. [Alcohol poisoning—use of ammonium acetate; cure].
 Journal de Chimie Médicale, de Pharmacie et de Toxicologie (Paris), 5: 128-129 (0 ref.), 1839.
 F – general – case hist. – DC (antidotal) – elect., water-bal. agents – *CAAAL-0 A-0907.

Case material is given concerning a 4-yr old child who became very ill after swallowing various alcoholic beverages—wine, brandy, rum, etc., comprising in all at least 2 oz of drink. The author found the child in a profound coma. Treatment was initiated with ammonium acetate (one grain) in a spoonful of sweetened water. A mustard poultice was applied to pelvic extremities, and chest and cold compresses were applied to the entire cranial surface, followed 1/2 hr later by a further application of ammonium acetate (1/2 grain). 1 hr after the initial administration, the child began to move. Ammonium acetate (1/2 grain) was continued up to a dose of 3 grains. Gradually, the intoxication receded, and the child completely recovered.

845. Marshall, E. K., Jr., Walzl, E. M., and LeMessurier, D. H.
 PICROTOXIN AS A RESPIRATORY STIMULANT.
 J. Pharmacol. Exp. Ther. (Baltimore), 60: 472-486 (15 ref.), 1937.
 E – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – cardiovasc. – respir. –
 stimulants – *CAAAL-0 A-0908.

In experiments with dogs and cats, picrotoxin (0.2-0.7 mg/kg iv) was found very effective in reinstating respiration which had failed, after phenobarbital administration, from carotid sinus inactivation, and from overdosage with chlorbutanol, paraldehyde, or “avertin fluid”. It was found less effective against overdosage with urethane, and useless against overdosage with ethyl alcohol (4 cats—1, 3,

or 4 doses of 0.5-2.0 mg alcohol/kg). Against phenobarbital- or chlorbutanol-induced respiratory failure, strychnine was ineffective, coramine was only slightly better, and metrazol was frequently effective in large dosage, but was inferior to picrotoxin.

846. Martin, W. B., Dyke, L. H., Jr., Coddington, F. L., and Snell, A. M.
**CARBON TETRACHLORIDE POISONING: A REPORT OF ONE CASE WITH
 NECROPSY AND ONE NONFATAL CASE WITH CLINICAL LABORATORY STUDIES.**
 Ann. Intern. Med. (Philadelphia), 25: 488-497 (23 ref.), 1946.
 E – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – humans – blood comp.,
 sites, lymph – cardiovasc. – G.I. tract – liver, kidney – respir. – anti-infectants – *CAAAL-0
 A-0909.

2 cases (1 fatal), of carbon tetrachloride (CCl_4) poisoning are reported. In 1 case, the patient, a heavy drinker, had been exposed to CCl_4 vapours daily for 6 months; the poisoning symptoms were precipitated by exposure to CCl_4 cleaning fluid vapours in a closed car. He died 8 days after hospitalization. In the second case, a man cleaned with a pint-container of CCl_4 for a period of 1 hr in a closed compartment. In the following 24 hr, he allegedly ingested alcohol. 48 hr after exposure, poisoning symptoms appeared and he was hospitalized. In both cases, the author considers alcohol to have been a probable adjuvant to the CCl_4 toxicity.

847. Martin du Pan, R.
**LA MODIFICATION DE LA RÉSORPTION DE L'ALCOOL APRÈS INGESTION
 D'HUILE DE PARAFFINE OU D'HUILE D'OLIVE.** [Modification of alcohol absorption after
 ingestion of paraffin oil or olive oil].
 Rev. Med. Suisse Rom. (Lausanne), 61(9): 537-549 (35 ref.), 1941.
 F – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo
 – blood lev. – G.I. tract – gastrointest. agents – *CAAAL-3678-A1 A-0910.

19 healthy young men received 1/2 l of wine (containing 48 g of absolute alcohol), and the blood alcohol concentration (BAC) was determined. The next day, they received the same amount of alcohol and 2 soup spoonsful of either olive oil or of paraffin oil. After 1/2 hr, the average BAC was 17% less with olive oil, and 2% less with paraffin oil. After 3 hr, the average BAC was 16% higher with olive oil, and 8% higher with paraffin oil. The highest concentrations were the same with or without the oils, so that intoxication was retarded but not diminished. The difference between the effects of olive oil and paraffin oil is explained by the fact that olive oil is absorbed by the gastric mucosa, whereas paraffin oil is not, and, since the mucosa cannot absorb the oil and the alcohol at the same time, "a sort of obstruction" of the cells occurs.

848. Mathieu, P., Serusclat, F., and Revol, L.
**INFLUENCE DE LA CHLORPROMAZINE SUR LE TAUX D'ALCOOL SANGUIN CHEZ
 LE LAPIN.** [Influence of chlorpromazine on the blood alcohol level in the rabbit].
 Société de Pharmacie de Lyon, Bulletin des Travaux (Lyon), 8(2): 79-83 (11 ref.), 1964.
 F – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in
 vivo – blood lev. – CNS – metab. proc. – sed., hypnot. – *CAAAL-0 A-1419.

The effects of chlorpromazine on the blood alcohol level (BAL) were studied in male and female rabbits. In 1 experiment, 20 mg/kg chlorpromazine hydrochloride po was administered simultaneously with 6 ml/kg aqueous ethanol sol (1:3 v/v) po. The BAL was slightly elevated to 164 mg/100 ml at 1 hr, as compared to 143 mg/100 ml for animals receiving ethanol only. In a second trial, 6 mg/kg was administered sc, followed by 6 ml/kg aqueous ethanol sol (1:3 v/v) po 90 min later. The BAL at 15 min was 170 mg/100 ml, compared to 134 mg/100 ml for the ethanol group. In a third experiment, identical to the second, except that a dosage of 6 ml/kg aqueous ethanol sol (1:6 v/v)

was given, the BAL at 15 min was 107 mg/100 ml, compared to 52 mg/100 ml for the ethanol group. The results appear to indicate a retarding of ethanol metabolism by chlorpromazine, probably by inhibition of alcohol dehydrogenase.

849. Mattison, J. B.

A CASE OF DOUBLE NARCOTIC ADDICTION—OPIUM AND ALCOHOL—IMBECILITY—RECOVERY.

Canada Lancet (Toronto), 17: 101-104 (3 ref.),

1884.

E – general – case hist. – conj. addict. – humans – CNS – senses – anti-infectants – gastrointest. agents – hallucinogens – stimulants – *CAAAL-0 A-0911.

The case of a woman who became addicted to morphine (several grains, 3-5 times/day) and alcohol (brandy, 12-16 oz/day) is reported. When she came under the author's care, "mentally she was a wreck. Delusions were prominent, and hallucinations of sight, sound and touch almost constant.... Her expression was idiotic; she was utterly unable to converse intelligibly, and her voice in speaking speedily sank to a whisper and was lost. In fact such mental ravages from opium we never met. Physically she was partially prostrated, pulse frequent and feeble, marked anorexia, furred tongue, and alvine torpor...." The unusually severe cerebral disturbances were aggravated by alcohol, although morphine was the main factor; the case was successfully treated with large doses of cannabis, the treatment employed almost exclusively by the author for opiate addiction. Another case, which the author declined, concerned a man with organic heart lesion who had for several years taken 10-20 grains of morphine sc, 60-90 grains of chloral, and 1-2 pints of whiskey daily. Although instances in which addiction to 1 narcotic leads to another are infrequent compared to those in which 1 alone is used, "the ruinous results exceed those of a single addiction, while the prospect of permanent cure is always less hopeful."

850. Mattison, J. B.

TRIPLE NARCOTIC ADDICTION: OPIUM, ALCOHOL, COCAINE.

Times and Register (Philadelphia), 21: 504 (0 ref.),

1890.

E – general – case hist. – conj. addict. – drug-dep. humans – cardiovasc. – CNS – G.I. tract – glands – liver, kidney – skel., muscle, skin – analg., antipyret. – anesthetics – hallucinogens – *CAAAL-0 A-1341.

Case histories of 2 physicians with simultaneous addiction to opium, alcohol, and cocaine are related. A young doctor, aged 34, was addicted to opium for 10 yr, followed by 16 months of triple narcotic addiction requiring 10-30 grains of morphine, 10-60 grains of cocaine, and 12-16 oz of rum/day. He lost 48 pounds during this time, and suffered from constipation and occasional attacks of nausea, vomiting, and anorexia. Loss of memory and mental hebetude were experienced. His skin was pallid and sallow, night sweating was profuse, and renal secretion was scanty. Sexual desire and power were negligible. A recovery was made after 6 weeks of treatment consisting of central galvanism, morning shower-baths, and full feeding. Another doctor, aged 46, developed, over a 4 yr-period, a triple narcotic addiction requiring 10 grains of morphine, 35 grains of cocaine, and 6 oz of rum/day. A liberal diet with cocoa and quinine constituted a successful 6 week therapy. *Cannabis indica* (Squibb's fluid extract) in 60 minim doses was the main hypnotic used in both cases.

851. Matussewitsch, I. S.

ZUR KLINIK DER BLEIVERGIFTUNG. [Clinical observations on lead poisoning].

Wien. Klin. Wschr. (Vienna), 41: 849-852 (0 ref.),

1928.

G – SEC – stat. surv. – humans – drug-dep. humans – blood comp., sites, lymph – cardiovasc. – CNS – G.I. tract – nerv. syst. – respir. – *CAAAL-0 A-1420.

Based on a study of 200 cases, the predisposing factors, early symptoms, and subjective and objective symptoms of industrial lead poisoning are discussed. Lead colic is described, with reference to

statistical data concerning the relationship of age, type of work, season, and duration of colic, to the frequency of occurrence. Chronic alcoholism is considered to be 1 of the predisposing factors. Using data from a survey of 815 workers, the author relates the severity of lead poisoning to the extent of alcohol consumption—abstinence, or light, moderate, or heavy drinking. The largest percentage of the most serious poisonings occurred in the heavy-drinking group, while the majority of workers who did not show any symptoms were in the abstinent category. It is concluded that the severity of poisoning symptoms is directly proportional to the degree of consumption of alcohol.

852. Maughs, G. M. B.

INCONSISTENCY IN THE USE OF ALCOHOLIC STIMULANTS WITH CHLOROFORM.

Medical Reporter (St. Louis), 1(7): 169-176 (0 ref.),

1866.

E – general – cross-tol. – DC (decrease) – humans – dose resp. – cardiovasc. – CNS – nerv. syst. – senses – anesthetics – *CAAAL-0 A-1342.

A former surgeon of the army of the Confederate States of America presents an argument against the use of alcoholic stimulants in cases of shock or hemorrhage, prior to chloroform anaesthesia and surgery. The argument is based on the opinion that chloroform, a powerful sedative, and alcohol, an active stimulant, have contradictory effects, and are therefore therapeutically incompatible. Chloroform lessens the tendency to hemorrhage by depressing the action of the heart and arteries, blunts the sensibility, and produces sleep; whereas alcohol increases the hemorrhagic tendency by increasing the tone of the vascular system, heightens sensibility, and prevents sleep. Since alcohol counteracts the chloroform, an overdose of the sedative drug is sometimes given in order to produce a given amount of anaesthesia, and death results by asphyxia or syncope, or by the formation of carbonic oxide. "Try it on a drunken man, and after the loss of much time and the administration of an unusual quantity of the anesthetic he becomes delirious, raves, gesticulates violently, vomits, and if by dint of perseverance you succeed in anesthetising him the probabilities are that he will be taken from the table a dead or dying man."

853. Mauriac

EMPOISONNEMENT PAR LA STRYCHNINE GUÉRI PAR LES ALCOOLIKES ET LE TANNIN. [Poisoning by strychnine cured by alcoholic beverages and tannin].

Société de Médecine et de Chirurgie de Bordeaux, Mémoires et Bulletins (Bordeaux), 377-379 (2 ref.),

1878.

F – general – case hist. – DC (antidotal) – humans – CNS – stimulants – *CAAAL-0 A-0912.

Case material is given concerning a young man who ingested 12 cg strychnine in a suicide attempt. 15 min later, he was seized with extreme tetanic convulsions. Treatment with 3 g tannic acid effected recovery. The other remedy in question consists of 2 dl rum and 3 g tannic acid. Following an emetic, another dose of rum is given, after which sleep, and then recovery, may be effected. The author refers to other articles which affirm the unquestionable antagonism between alcohol and strychnine.

854. Mayer, K., Martin, H., and Mallach, H. J.

MOTORISCHE NERVENLEITGESCHWINDIGKEIT UNTER DEM EINFLUSS VON ALKOHOL UND DIMETHYLSULFOXYD. [Velocity of motor impulse conduction under the influence of alcohol and dimethylsulfoxide].

Arzneimittelforschung (Aulendorf), 16: 1226-1228 (9 ref.),

1966.

G – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – species or sex diff. – G.I. tract – nerv. syst. – *CAAAL-0 B-0375.

In 46 persons, the velocity of motor impulse conduction (MIC) was measured on an empty stomach, after alcohol intake, and after dimethylsulfoxide (DMSO) plus alcohol. Variations with the time of day could be observed, but were insignificant. Specific differences between the 2 sexes were an-

ticipated, but could not be confirmed. Alcohol (0.75 g/kg) diminished the average MIC for approximately 6 meters/sec., whereas DMSO (50 mg/kg) plus alcohol diminished it for approximately 11.6 meters/sec. These changes were reversible, since the values tended to normalize with lowering alcohol concentration of the blood. Further investigation should clarify the manner in which alcohol and DMSO, alone or in combination, influence the body temperature, the carbon dioxide content of the blood, and the permeability of the cell membrane.

855. Mayer, R. M.
VERZÖGERTER ALKOHOLUMSATZ BEI LUMINALVERGIFTUNG. [Delayed alcohol metabolism in poisoning with luminal].
 Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 27: 80 (0 ref.), 1937.
 G – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – other drug lev. – metab. proc. – barbiturates – *CAAAL-0 A-0913.

A fatal case of drug poisoning is reported, concerning a man who ingested about 200-220 g absolute alcohol (as beer and distilled spirits) plus 11 tablets of luminal (0.3 g each), and was then given another 10 luminal tablets by his wife. Only after 24 hr was a physician called. The patient died a few hr after admission to hospital. The post mortem revealed, 40 hr after the last drink, a blood alcohol concentration of 1.3°/oo, and a urine concentration of 1.2°/oo. It is assumed that the highest blood alcohol concentration must have been 3 times as much. The wife was committed to trial for murder.

856. Mayrant, W.
ON THE USE OF ALCOHOL IN THE DISEASE PRODUCED BY THE BITE OF THE RATTLESNAKE.
 American Medical Recorder (Philadelphia), 6: 619-621 (1 ref.), 1823.
 E – general – case hist. – DC (unspec.) – humans – cardiovasc. – CNS – G.I. tract – nerv. syst. – skel., muscle, skin – *CAAAL-0 A-1421.

The use of alcohol in 3 cases of rattlesnake bite is presented. A negro male was bitten and found lying motionless and speechless, with locked jaws and a very feeble pulse. A teaspoonful of finely powdered red pepper was mixed with a glass of whiskey and poured down the victim's throat. After vomiting up 3 or 4 glassfuls of this mixture, 5 or 6 more glassfuls were kept down, and the pulse was observed to improve. With continued administration of alcohol and pepper, the man recovered after approximately 12 hr. Another man bitten by a rattlesnake recovered after consuming about a quart of alcohol mixed with green pepper over a 10-12 hr period. A third victim, whose experience was related afterwards to the author, is said to have recovered from a poisonous snake bite through having been in a state of intoxication at the time of the bite. It is stated that large doses of brandy and opium have also been successful in treating poisonous snake bites.

857. Mazaud, R., and Mafart, Y.
INTOXICATION PAR LE TÉTRACHLORURE DE CARBONE: ROLE AGGRAVANT DES TARES VISCÉRALES ET DE L'ÉTHYLISME. [Carbon tetrachloride poisoning: aggravating role of visceral disorders and ethylism].
 Société de Médecine Militaire Française, Bulletin (Paris), 48: 174-176 (5 ref.), 1954.
 F – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – liver, kidney – anti-infectants – *CAAAL-7422-E8 A-0914.

2 French seamen, 1 an alcoholic, repaired and cleaned carbon tetrachloride (CCl₄) fire extinguishers for 1 hr in a poorly ventilated room. Both developed symptoms of CCl₄ poisoning; 1 man recovered in a day, but the alcoholic became extremely ill, and required extensive treatment for about 3 weeks. At the time of exposure, the latter man had not yet eliminated the alcohol consumed at his noon meal. The author discusses other research and reported cases concerning the effect of alcohol on CCl₄.

toxicity. He concludes that personnel involved in work with the risk of CCl_4 exposure must neither be chronic alcoholics, nor ingest alcohol prior to such exposure, and that work with the chemical should take place after as long a time interval as possible between meals, since, in France, appreciable amounts of alcohol are frequently consumed at mealtime.

858. Mazzucchelli, B., and Guarneri, A.

RICERCHE SPERIMENTALI SULLE VARIAZIONI DELL'ASSORBIMENTO E DELL'ELIMINAZIONE ALCOOLICA DOPO ALCUNI TRATTAMENTI USATI IN CHIRURGIA. [Experimental investigations on absorption and elimination of alcohol after some treatments used in surgery].

Minerva Medicoleg. (Turin), 86: 189-192 (6 ref.),

1966.

I – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – analg., antipyret. – sed., hypnot. – tranquilizers – *CAAAL-0 B-0376.

15 female rabbits were given 0.15 g/kg absolute alcohol sol (1:2 ratio of dilution) iv, in combination with the following: a mixture of fargan, mefeldina, and largactil; 2-2.5 cc thiopentone; or 30 cc polyvinylpyrrolidone (subtosan). The blood alcohol curve was significantly altered by all 3 combinations. The fargan-mefeldina-largactil preanesthesia produced a mean retardation of the alcohol elimination rate of up to 31% in the first 16 min. The thiopentone retarded elimination by up to 51% in 25.5 min, and subtosan increased it by up to 16% in 7.8 min.

859. Meerhoff, A., and Meerhoff, W.

TRATAMIENTO DE LA AMBLIOPIA ALCOHOLO-TABÁGICA CON ALTAS DOSIS DE ESTRICNINA. [The treatment of alcohol-tobacco amblyopia with large doses of strychnine].

Universidad de Montevideo, Facultad de Medicina, Anales (Montevideo), 14: 114-119 (1 ref.),

1929.

Sp – FS – general – case hist. – DC (antidotal) – humans – senses – stimulants – *CAAAL-0

A-0915.

2 patients suffering from alcohol-tobacco amblyopia were treated with high doses of strychnine, which were increased as the tolerance of the patients developed; 3 mg/day sc were given on the first day, and the dosage was gradually increased to 16 mg/day. The condition was rapidly cured in each case. The desire to drink disappeared, and the strychnine substantially stimulated the appetite; due to the latter factor, the nutritional deficiencies were quickly overcome and the health of the patients improved accordingly. The authors feel that large strychnine doses are to be much preferred to the customary small therapeutic dosage.

860. Meerhoff, A., and Meerhoff, W.

TRATAMIENTO DE LA AMBLIOPIA ALCOHOL-TABÁQUICA CON ALTAS DOSIS DE ESTRICNINA. [The treatment of alcohol-tobacco amblyopia with large doses of strychnine].

Semana Médica (Buenos Aires), 36(2): 55-56 (1 ref.),

1929.

Sp – general – case hist. – DC (antidotal) – humans – senses – stimulants – *CAAAL-0 A-0916.

2 patients suffering from alcohol-tobacco amblyopia were treated with high doses of strychnine, which were increased as the tolerance of the patients developed; 3 mg/day sc were given on the first day, and the dosage was gradually increased to 16 mg/day. The condition was rapidly cured in each case. The desire to drink disappeared, and the strychnine substantially stimulated the appetite; due to the latter factor, the nutritional deficiencies were quickly overcome and the health of the patients improved accordingly. The authors feel that large strychnine doses are to be much preferred to the customary small therapeutic dosage.

861. Meerlo, J. A. M.
VARIABLE INDIVIDUAL TOLERANCE FOR ALCOHOL AND DRUGS.
 Postgrad. Med. (Minneapolis), 22: 583-590 (3 ref.), 1957.
 E – SEC – general – case hist. – cross-tol. – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – drug dep. humans – psychol. perform. – CNS – liver, kidney – respir. – autotoxics – barbiturates – stimulants – *CAAAL-8241-Z14 A-0917.

Factors influencing tolerance and intolerance to alcohol and drugs are discussed. The problems involved in putting the alcoholic under anesthesia, and in administering drugs to such persons, are discussed. Barbiturates and morphine, in particular, change the tolerance for alcohol. The author was forced to forbid the use of alcohol by patients undergoing treatment with increasing doses of atropine for post-encephalitic parkinsonism, because acute alcohol intoxication could result in either acute alcoholic intolerance, or an atropine intolerance. The barbiturates can also lead to cumulative action and midbrain damage through the uncontrolled use. Cerebral disease, oxygen lack, alcohol allergy, dangers of withdrawal and abstinence, and pathological and hereditary intolerance are discussed, and illustrative cases are cited.

862. Melville, K. I., Joron, G., and Douglas, D.
COMBINED ALCOHOL AND GLUTETHIMIDE OR SECOBARBITAL CENTRAL NERVOUS SYSTEM POTENTIATION.
 Canadian Federation of Biological Societies, Proceedings (Montreal), 5: 53-54 (0 ref.), 1962.
 E – abst. – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – CNS – respir. – barbiturates – sed., hypnot. – *CAAAL-0 A-1343.

The combined effects of alcohol and 2 sleep-inducing drugs were studied in dogs. The following were administered by stomach tube: 1) 1 ml 25% alcohol (A)/kg/hr in 3 doses, 2) 200-500 mg glutethimide(G)/kg in water, 3) G combined with the third dose of A, 4) 25-500 mg secobarbital sodium (S)/kg in water, and 5) S plus A. It was found that A produced mild ataxia, with recovery in 6-12 hr. 300 mg G/kg produced variable increasing CNS depression, with recovery in 24-48 hr, and 500 mg G/kg produced death in 24-48 hr. A plus 200-300 mg G/kg resulted in collapse, coma with respiratory depression, and death within 72 hr; A plus 500 mg G/kg led to collapse within 30 min, and early or delayed death. 25 mg S/kg induced CNS depression and recovery in 12 hr, but 50 and 100 mg S/kg resulted in death within 10-94 min. A plus 25 mg S/kg produced collapse for 6 hr, with recovery in 24 hr (or death in 1 experiment). There were no differences in blood alcohol or barbiturate levels among the various groups. It is concluded that the respiratory depressant and toxic effects of these hypnotic agents are potentiated in the presence of moderate alcohol depression.

863. Melville, K. I., Joron, G. E., and Douglas, D.
TOXIC AND DEPRESSANT EFFECTS OF ALCOHOL GIVEN ORALLY IN COMBINATION WITH GLUTETHIMIDE OR SECOBARBITAL.
 Toxic. Appl. Pharmacol. (New York), 9: 363-375 (20 ref.), 1966.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – absorp., distrib., stor. – CNS – barbiturates – sed., hypnot. – *CAAAL-12511-D2 B-0377.

General central depressant and toxicological effects were compared in dogs after the following were administered po: alcohol alone (3 doses of 1 ml/kg 50% ethanol, 1 hr apart), glutethimide (200, 300, or 500 mg/kg, alone or in combination with the third dose of alcohol), or secobarbital sodium (25 mg/kg, alone or in combination with the third dose of alcohol). Blood alcohol and blood barbiturate levels were determined at hourly intervals, and blood glutethimide levels determined at 6-hr intervals. The onset, intensity, and duration of the central depressant effects and toxicity of glutethimide and secobarbital were strikingly enhanced by alcohol in all experiments. Blood alcohol level changes up to 6 hr were not significantly different after alcohol, drug, or combined administrations, but, after

6 hr, the blood alcohol levels declined significantly in tests with the combined administrations. Blood glutethimide and barbiturate levels also decreased significantly in the experiments with added alcohol. It is postulated that a rapidly increased absorption of glutethimide or secobarbital, due to the presence of alcohol in the stomach, might be one of the factors involved in the enhanced CNS depression induced by the combinations.

864. Mendelson, J., Wexler, D., Leiderman, P. H., and Solomon, P.

A STUDY OF ADDICTION TO NONETHYL ALCOHOLS AND OTHER POISONOUS COMPOUNDS.

Quart. J. Stud. Alcohol (New Haven), 18: 561-580 (28 ref.), 1957.
E – general – review – case hist. – cross-tol. – DC (decrease) – drug-dep. humans – acid-base, blood pH, elect. – blood comp., sites, lymph – liver, kidney – alcohols – *CAAAL-8536-L4 A-1422.

Case histories are presented concerning 9 adult alcoholics, who, in addition to addiction to ethanol for 19-40 yr, were chronic imbibers of toxic alcohols, such as methanol, isopropyl alcohol, paraldehyde, and methyl salicylate, for 10-40 yr. All patients were hospitalized following an episode of heavy drinking of ethanol plus 1 or more toxic substances; 5 men were in delirium tremens, 2 had alcoholic hallucinosis, 1 was comatose (the blood paraldehyde level was 100 mg/100 ml in serum), and 1 was acutely intoxicated. All but 1 had evidence of hepatomegaly, but none were jaundiced or had ascites or splenomegaly; 6 had normal liver function tests, and 3 tested only slightly abnormal. None had any permanent kidney damage, and electrolytes were remarkable for their lack of severity—in 6 cases, sodium, potassium, and chloride were within normal limits. Renal studies were within normal ranges. All patients responded well to treatment, and were discharged as recovered in 5 days to 4 weeks, at which time there was no evidence of neurological deficit, except in 2 patients with peripheral neuropathy. The unexpected rapid and complete recovery, the speed of metabolism of toxic substances, the great immunity of the body organs, and the general good health of the alcoholics, despite the fact that they habitually ingested amounts, "that might have been fatal to a dozen ordinary men," are discussed, and explanations are offered.

865. Menninger-Lerchenthal, E.

KRITIK DER ALKOHOLERNÜCHTERUNGSVERSUCHE. [Critical evaluation of sobering-up methods].

Wien. Klin. Wschr. (Vienna), 72(23): 419-422 (44 ref.), 1960.
G – review – DC (decrease) – DC (unchanged) – humans – other drug lev. – nerv. syst. – amphetamines – anesthetics – elect., water-bal. agents – hormones, hormone antag. – stimulants – *CAAAL-9896-N14 A-0918.

The literature on the possibility of reducing the intoxicating effects of alcohol in man by drugs, stress, cold showers, or caffeine-containing beverages is reviewed. Stimulation of the nervous system, may, if the intoxication is not too severe, give an illusion of mitigating the effects of alcohol. This pseudo-sobriety was found to be short-lived, however, as long as there is alcohol in the brain. The artificial sobering-up methods are, according to the author, unrealistic therapeutic means to treat alcohol intoxication.

866. Meyer, A.

ANTABUSÄHNLICHE WIRKUNG VON IRGAPYRIN. [Antabuse-like effect of irgapyrin].

Schweiz. Med. Wschr. (Basel), 85: 94 (1 ref.), 1955.
G – general – DC (sensit.) – humans – cardiovasc. – metab. proc. – *CAAAL-7221-A15 A-1423.

In an open letter, the author comments on the article by E. Läubli (Schweiz. Med. Wschr., 84: 1281-1283, 1954), concerning the disulfiram-like properties of irgapyrin (aminophenazone + phenyl-

butazone). He points out that not only pyrazole derivatives have this effect. Egressin, which is not chemically related to irgapyrin or disulfiram, produces an intolerance reaction after either alcohol or nicotine consumption. Also, heavy smoking increases intolerance to alcohol. The fact that alcoholics are susceptible to pellagra indicates the possibility that there is a link with the PP factor, a coenzyme of diphosphopyridine nucleotide, which promotes acetaldehyde decomposition. The disulfiram treatment is a psychological one, creating Pavlovian conditioned reflexes. It accomplishes the same goal as prolonged institutionalization, without the enormous cost of the latter. Lauppi replies that the effects of smoking on alcohol tolerance were in fact examined, but the individual differences which were found were so great that no general conclusions could be drawn. As to conditioned reflex, apomorphine can be used, but, in the case of disulfiram, the symptoms are due to actual toxic products of the disulfiram-alcohol interaction.

867. Meyers, D. B., Kanyuck, D. O., and Anderson, R. C.
EFFECT OF CHRONIC NORTRIPTYLINE PRETREATMENT ON THE ACUTE TOXICITY OF VARIOUS MEDICINAL AGENTS IN RATS.
 J. Pharm. Sci. (Washington), 55: 1317-1318 (10 ref.), 1966.
 E – SEC – exp. cont. – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – CNS – antidepressants – *CAAAL-12598-D2 B-0378.

Groups of rats received a diet containing nortriptyline hydrochloride (0.4%) or amitriptyline hydrochloride (0.04%) for 11-15 days. Cerebral depressants, CNS stimulants, autonomic agents, and various other agents were then administered ip every 30 min until death ensued. A cumulative lethal dose (CLD) for each agent was determined by multiplying the amount of drug per dose (mg/kg) by the number of doses required for death. An index of interaction (II) was then established by dividing the geometric mean of CLD values for the controls by the geometric means of the CLD in each of the pretreated groups. An II significantly larger than 1.00 indicated synergism, and an II significantly less than 1.00 indicated antagonism. When ethanol was given after nortriptyline or amitriptyline pretreatment, the II was .95 and 1.10, respectively. Thus the study did not show any significant effect on ethanol toxicity by either amitriptyline or nortriptyline, and failed to confirm clinical reports that the tricyclic antidepressives dangerously potentiate the effects of alcohol.

868. Mezey, K.
DIE WIRKUNG DES CORAMINS UND DES CALCIO-CORAMINS AM STARLINGSCHEN HERZ-LUNGENPRAPARAT. [The action of coramine and calcio-coramine on the heart-lung-preparation of Starling].
 Naunyn-Schmiedebergs Archiv fur Experimentelle Pathologie und Pharmakologie (Berlin), 177: 235-247 (31 ref.), 1935.
 G – exp. – DC (decrease) – mammals – acute admin. – in vivo – cardiovasc. – stimulants – *CAAAL-1183-D2 A-0919.

The effects of coramine and calcio-coramine were investigated in 40 heart-lung preparations. Coramine, in concentrations of 1:3750 to 1:3000, increased the minute-volume and the arterial pressure. When alcohol in a concentration of 4:150 was added to the venous reservoir, the arterial pressure went down, the venous pressure increased, and the minute-volume decreased; if coramine (1:3000) was then added, the alcohol-induced fall of arterial pressure could only be stopped for 20 to 30 seconds.

869. Michel, E.
UBER DIE MEDIKAMENTOS BEDINGTEN ABNORMEN ALKOHOL-WIRKUNGEN BEIM KRAFTFAHRER. [Abnormal effects of alcohol in the driver caused by drugs].
 Dissertation, Medical Faculty of the University of Frankfurt, West Germany, 38 pp. (61 ref.), 1959.
 G – general – stat. surv. – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans

– blood lev. – other drug lev. – analg., antipyret. – autocoids – sed., hypnot. – tranquilizers – *CAAAL-0 A-0920.

The literature of alcohol-drug interactions is reviewed. From the files of the Institute of Forensic Medicine in Frankfurt, 10 court cases in which interaction between alcohol and drugs was established are discussed. The various theories of interaction of alcohol with hypnotics, analgesics, antihistamines, antipyretics, sedatives, and tranquilizers are presented. It is suggested that, in every accident in which the driver is found to be intoxicated, blood and urine tests should also be analyzed for the presence of drugs.

870. Mickat, K.

ÜBER DIE WIRKUNGSWEISE EINER KOMBINATION VON NATRIUM-DIAETHYLBARBITURAT (= VERONAL-NATRIUM) UND ALKOHOLGABEN MIT GLEICHZEITIGEN UNTERSUCHUNGEN ÜBER DIE AUSSCHIEDUNG DER BARBITURSÄURE. [The mechanism of action of a combination of sodium diethylbarbiturate (veronal sodium), with subsequent investigation of the excretion of barbituric acid].

Dissertation, Medical Faculty of the University of Hamburg, West Germany, 40 pp. (21 ref.),

1964.

G – exp. cont. – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – acute admin. – in vivo – blood lev. – other drug lev. – psychol. perform. – barbiturates – *CAAAL-0 A-0921.

The combined effect of barbital sodium and alcohol was studied in relation to driving performance, and, secondly, an attempt was made to determine the practical value of the urine test for forensic purposes. 10 students with a blood alcohol concentration of about 1.2°/oo ingested po barbital sodium in dosages up to 500 mg. Various psychological tests were applied. 250 mg of barbital sodium, ingested at a blood level of about 0.8°/oo, was found to have an additive effect, resulting in further impairment of driving performance. The urine analysis showed different values with different subjects, although drug dosage and ingestion time were the same, thus proving that urine-barbiturate values do not permit definite conclusions as to dosage ingested and time of ingestion.

871. Mikkonen, H.

COMPARATIVE CLINICAL STUDY OF ALCOHOLICS VOLUNTARILY SEEKING TREATMENT: PRELIMINARY REPORT.

Acta Neurol. Belg. (Brussels), 67(Suppl.): 73-78 (6 ref.),

1967.

E – DS – FS – GS – exp. comp. – DC (antidotal) – drug-dep. humans – acute admin. – chronic admin. – in vivo – CNS – tranquilizers – *CAAAL-0 B-0379.

In a double-blind study, the therapeutic value and possible side effects of dixyrazine, a neuroleptic drug, and hydroxyzine, a tranquilizer, were compared in 120 male alcoholics, 99 of whom were intoxicated when hospitalized. For intoxication, hydroxyzine (300 mg/kg for 2 days) and dixyrazine (60 mg/day for 2 days) had a slight therapeutic effect in 10 and 12 patients, respectively, a good effect in 33 and 23, and a very good effect in 6 and 15; in 21 patients treated for hangover, hydroxyzine and dixyrazine had a slight effect in 3 and 1, respectively, and a good effect in 8 and 5. No serious side effects were observed, and the anxiolytic effect was pronounced. The therapeutic action of dixyrazine was shown to be more effective than that of hydroxyzine. Generally, the acute condition was overcome after 2 days, and treatment was continued with thioridazine.

872. Mikkonen, H.

COMPARATIVE CLINICAL STUDY OF ALCOHOLICS VOLUNTARILY SEEKING TREATMENT (A PRELIMINARY REPORT).

Int. J. Neuropsychiat. (Chicago), 3(5): 418-421 (6 ref.),

1967.

E – exp. comp. – DC (antidotal) – drug-dep. humans – acute admin. – chronic admin. – in vivo – CNS – tranquilizers – *CAAAL-0 B-0380.

In a double-blind study, the therapeutic value and possible side effects of dixyrazine, a neuroleptic drug, and hydroxyzine, a tranquilizer, were compared in 120 male alcoholics, 99 of whom were intoxicated when hospitalized. For intoxication, hydroxyzine (300 mg/day for 2 days) and dixyrazine (60 mg/day for 2 days) had a slight therapeutic effect in 10 and 12 patients, respectively, a good effect in 33 and 23, and a very good effect in 6 and 15; in 21 patients treated for hangover, hydroxyzine and dixyrazine had a slight effect in 3 and 1, respectively, and a good effect in 8 and 5. No serious side effects were observed, and the anxiolytic effect was pronounced. The therapeutic action of dixyrazine was shown to be more effective than that of hydroxyzine. Generally, the acute condition was overcome after 2 days, and treatment was continued with thioridazine.

873. Miller, A. I., D'Agostino, A., and Minsky, R.
EFFECTS OF COMBINED CHLORDIAZEPOXIDE AND ALCOHOL IN MAN.
Quart. J. Stud. Alcohol (New Haven), 24(1): 9-13 (7 ref.), 1963.
E – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – mot. perform.
– psychol. perform. – cardiovasc. – CNS – tranquilizers – *CAAAL-10880-D1 A-0922.

8 human subjects underwent 2 trials with chlordiazepoxide and 2 with placebo; the drug trials were 1 week apart, followed 9 weeks later by the placebo trials, which were 1 week apart. For 4 days, 10 mg chlordiazepoxide or placebo were taken 4 times/day. On the 4th day, 4 oz of scotch whiskey were consumed by each subject, over a 0.5 hr interval. Blood samples were taken, and blood pressure, pulse, respiration, digit symbol performance, and behavioural determinations were made 0.5, 1.5, 3, and 4.5 hr after alcohol. The data showed no qualitative difference between the effects of chlordiazepoxide and placebo in social behaviour produced by alcohol consumption, in physiological measurements, or in performance in the Wechsler digit symbol test. The mean values of blood alcohol were slightly higher in the placebo-alcohol tests at 3 and 4.5 hr after alcohol, but the differences were not statistically significant.

874. Miller, J. G., and Uhr, L.
BEHAVIORAL TOXICITY AS MEASURED BY TESTS OF SIMULATED DRIVING AND OF VISION.
In: Miller, James G., et al., eds. *Drugs and behavior*. New York: John Wiley and Sons, pp. 326-329 (9 ref.), 1960.
E – SEC – exp. cont. – exp. comp. – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – mot. perform. – senses – tranquilizers – *CAAAL-0 A-0923.

The acute effects in normal subjects of double the normal doses of meprobamate, dextro-amphetamine sulfate, meprobamate plus alcohol (2 oz, 80 proof), and alcohol alone were tested. Accuracy, speed, judgment, and reaction-time were tested on a driver-trainer, and visual functions were tested with the Bausch and Lomb Ortho-rater. There was some evidence of unsteadiness under alcohol. No behavioural toxic effects were found with the other 3 treatments, as compared with placebo controls. Chronic meprobamate, prochlorperazine, and tranquil (a triple-bromide preparation) effects were also determined.

875. Miller, J. G.
OBJECTIVE MEASUREMENTS OF THE EFFECTS OF DRUGS ON DRIVER BEHAVIOR.
J.A.M.A. (Chicago), 179(12): 940-943 (9 ref.), 1962.
E – SEC – exp. cont. – exp. comp. – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – mot. perform. – senses – tranquilizers – *CAAAL-0 A-0035.

The acute effects on normal subjects of double the normal doses of meprobamate, dextro-amphetamine sulfate, meprobamate plus alcohol (2 oz, 80 proof), and alcohol alone were tested. Accuracy,

speed, judgement, and reaction-time were tested on a driver-trainer, and visual functions were tested with the Bausch and Lomb Ortho-rater. There was some evidence of unsteadiness under alcohol. No behavioural toxic effects were found with the other 3 treatments, as compared with placebo controls. Chronic meprobamate, prochlorperazine, tranquil (a triple-bromide preparation), chlordiazepoxide, mebutamate, and phenobarbital effects, and acute effects of benactyzine, were also determined.

876. Miller, M. M.

AMPHETAMINE SULFATE IN ABORTING THE ACUTE ALCOHOLIC CYCLE.

Amer. J. Psychiat. (Hanover), 100: 800-802 (6 ref.), 1944.
 E – exp. cont. – DC (antidotal) – drug-dep. humans – acute admin. – chronic admin. – in vivo – CNS
 – senses – amphetamines – barbiturates – *CAAAL-4085-M21 A-0924.

56 non-psychotic, chronic alcoholics were treated with 10 mg amphetamine sulfate 2 times/day, 1/4 g luminal/day, and 30-40 mg thiamine chloride/day. 8 control patients received placebo in place of amphetamine. It was found that the acute drinking cycle was interrupted in 49 of the 56 patients, with subsequent periods of abstinence ranging from 1 to 18 months or longer. Single doses of amphetamine produced marked analeptic effects in patients who were still semi-stuporous; within 30-120 min they were partially or completely awakened, and there was a notable improvement in mood, physical well-being, critical reasoning, and activity drive. Patients who were not stuporous were usually relieved of the post-intoxication symptom complex, and stated that they could concentrate better. None of the control group exhibited the above effects, and continued drinking.

877. Miller, M. M.

COMBINED USE OF ETHYL ALCOHOL AND AMOBARBITAL (AMYTAL) SODIUM FOR AMBULATORY NARCOANALYSIS.

A.M.A. Archives of Neurology and Psychiatry (Chicago), 67: 620-624 (10 ref.), 1952.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – psychot. humans – acute admin. – in vivo – blood lev. – psychol. perform. – cardiovasc. – CNS – respir. – barbiturates – elect., water-bal. agents
 – sed., hypnot. – *CAAAL-6124-V35 A-0925.

Amobarbital sodium, 120 to 230 mg in 20 to 25 cc of a 10 to 15% sol of ethanol, was administered iv to 50 patients with nervous disorders. The effects on verbal productivity, emotional discharge, dissipation of drowsiness, and gross clinical ataxia were determined. In contrast to amobarbital sodium or thiopental sodium, the alcohol-amobarbital sodium sol did not appear to depress circulation or respiration, and in most patients it appeared initially to stimulate blood pressure, pulse vol, and pulse rate, and to deepen respiration, although the respiration rate was slightly lowered. The recovery of conscious awareness and sufficient orientation occurred sooner with alcohol-amobarbital sodium, and a more accurate control of the degree of narcosis was effected. In the author's opinion, alcohol-barbiturate synergism rests on the fact that alcohol has a considerably higher degree of solubility in lipids, and acts as a vehicle for barbiturates, carrying considerably more barbiturate into the brain. Aqueous sol are far less soluble in lipids, and hence penetrate the brain more slowly and to a slighter degree (Meyer-Overton theory of narcosis).

878. Milner, G.

AMITRIPTYLINE-POTENTIATION OF ALCOHOL.

Lancet (London), 1: 222-223 (0 ref.), 1967.
 E – exp. cont. – exp. comp. – general – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
 – in vivo – CNS – antidepressants – *CAAAL-0 B-0381.

In a letter to the editor, the author describes experiments with mice given alcohol and the antidepressants amitriptyline or trimipramine. In 1 series of experiments, the effect of 50 mg/kg amitriptyline plus 25 ml/kg 25% ethanol was compared with alcohol-placebo and drug-placebo; over a period of

5 hr, the length of loss of righting reflex was 3 times greater in the drug-alcohol group than in the alcohol-placebo group (probability < 0.001), and the drug-placebo group showed no loss of righting reflex. 70 of the 270 mice treated with drug-alcohol died. Similar tests were conducted on 220 mice, using 30 mg/kg amitriptyline, and potentiation of alcohol was significant (probability < 0.01); of 110 mice given drug plus alcohol, 6 died. Trimipramine was also found to potentiate alcohol significantly, but without causing as high a mortality rate.

879. Milner, G.

CUMULATIVE LETHAL DOSE OF ALCOHOL IN MICE GIVEN AMITRIPTYLINE.

J. Pharm. Sci. (Washington), 57(11): 2005-2006 (8 ref.), 1968.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – antidepressants – *CAAAL-0 B-0382.

The interaction effects of amitriptyline and alcohol were evaluated using albino mice. Of 6 groups of 10 mice, 3 were given 50 mg/kg amitriptyline, and 3 received a placebo sol. The mice were then given an alcohol sol at 2 hr intervals—4 groups received 12.5 ml/kg 25% alcohol doses, and 2 groups received doses of 10 ml/kg 25% alcohol. Loss of righting reflex (LRR) and time of death were observed. For the mice given 10 ml/kg 25% alcohol, the average number of alcohol doses required to cause death was 4.0, compared to 6.1 for placebo controls. Little difference between the drug-alcohol and control groups was noted in the first hr, when the cumulative LRR was plotted, but, as the experiment progressed, the difference between the groups was increasingly marked.

880. Milner, G.

INTERACTION OF PSYCHOTROPIC DRUGS AND ALCOHOL.

Australian and New Zealand Journal of Psychiatry (Victoria), 2: 65-66 (9 ref.), 1968.

E – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – CNS – hallucinogens – tranquilizers – *CAAAL-0 B-0383.

The work of various authors on the interaction of various psychotropic drugs and alcohol with respect to driving is reviewed. One author has established that chlorpromazine has a supplementary, and possibly potentiating, effect on the impairment of coordination and judgment produced by alcohol. Chlordiazepoxide was found to potentiate alcohol in laboratory animals, but this interaction was not confirmed in human laboratory tests. However, during a 90-day driving test, subjects who were administered chlordiazepoxide had 10 times more accidents than control groups. Another author reported that death of subjects with a blood alcohol level of only 0.1% may result from as little as 0.5 mg% of barbiturate.

881. Milner, G.

MODIFIED CONFINEMENT MOTOR ACTIVITY TEST FOR USE IN MICE.

J. Pharm. Sci. (Washington), 57(11): 1900-1902 (7 ref.), 1968.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – mot. perform. – antidepressants – *CAAAL-0 B-0384.

The modification of confinement motor activity (CMA) test for use with mice is described. Its use in quantitating the effect of drugs on the motor activity in mice is illustrated by experiments with amitriptyline and alcohol given by stomach tube. Amitriptyline (10 mg/kg) was found significantly to increase CMA when compared with control groups. Alcohol (25 ml/kg, 10% sol) depressed CMA in mice, but not to a statistically significant extent. When the amitriptyline and alcohol were given together, a significant depression of CMA was recorded. A positive joint action is suggested—amitriptyline, when taken by man, may add to the effects of alcohol.

882. Milner, G.
THE EFFECT OF ANTIDEPRESSANTS AND "TRANQUILLIZERS" ON THE RESPONSE OF MICE TO ETHANOL.
 Brit. J. Pharmacol. (London), 34: 370-376 (15 ref.), 1968.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged)
 – mammals – acute admin. – in vivo – CNS – liver, kidney – antidepressants – *CAAAL-0
 B-0385.

The interactions of amitriptyline, trimipramine, imipramine, nortriptyline, desipramine, thioridazine, phenelzine, methylphenidate, chlorpromazine, trifluoperazine, phenobarbitone, and diazepam (doses ranged from 2 mg/kg to 50 mg/kg po) with an oral dose of ethanol (25 ml/kg of a 25% sol) were tested in 3140 mice. The parameters measured were: length of loss of righting reflexes, continuous coma, number of animals remaining in coma 12 hr after dose administration, and changes in toxicity. Imipramine caused no significant changes in the effects of ethanol. Methylphenidate and desipramine protected the mice against ethanol-induced coma. All other drugs induced statistically significant potentiation of the depressant and toxic effects of ethanol.

883. Milner, G.
CHLORPROMAZINE AND THIORIDAZINE—A COMPARISON OF THEIR EFFECTS, PARTICULARLY IN POTENTIATING ALCOHOL.
 Pakistan Medical Review (Karachi), 3: 19-24 (11 ref.), 1969.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – mot. perform. – CNS – tranquilizers – *CAAAL-0
 B-0386.

The effects of thioridazine (5-10 mg/kg) and chlorpromazine (10 mg/kg), alone and in combination with 25 mg/kg 20% alcohol sol, on the confinement motor activity (CMA) of albino mice were tested. Thioridazine and chlorpromazine added to and prolonged the depression of CMA caused by alcohol, thioridazine being much less potent in this respect than chlorpromazine. It is suggested that, because of the relatively low incidence of side effects from thioridazine, and the possibility that it is less potent in adding to the sedative effects of alcohol than chlorpromazine, it may be more suitable for use with out-patients.

884. Milner, G.
DRINKING AND DRIVING IN 753 GENERAL PRACTICE AND PSYCHIATRIC PATIENTS ON PSYCHOTROPIC DRUGS.
 Brit. J. Psychiat. (London), 115(518): 99-100 (11 ref.), 1969.
 E – stat. surv. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – psychot. humans – CNS – hallucinogens – *CAAAL-0
 B-0387.

A survey was made of 4,584 patients under the care of a number of general practitioners and psychiatrists, to determine the types of drugs taken by these patients. Psychotropic drugs were prescribed to 753 of the patients; 85% of the men took alcohol, 66% were licensed drivers, and 57% were exposed to the risk of drinking and driving while taking the drugs. The corresponding figures for women were 71%, 42% and 35%. It is concluded that, since psychotropic drugs may potentiate the effects of alcohol, special hazards may be presented to the drinking driver.

885. Milner, G.
GASTRO-INTESTINAL SIDE EFFECTS AND PSYCHOTROPIC DRUGS.
 Med. J. Aust. (Sydney), 2: 153-155 (20 ref.), 1969.
 E – SEC – general – DC (add., infra-add., unspec. incr.) – humans – CNS – G.I. tract – antidepressants – tranquilizers – *CAAAL-0
 B-0574.

The general problem of gastro-intestinal side effects from the use of psychotropic drugs is discussed. Owing to the large numbers of psychotropic drugs used, many side effects, adverse or otherwise, occur as a result of taking not only 1, but more than 1 agent at a time. Chlorpromazine alone may inhibit hormone release, inhibit enzymes, or alter metabolism. Atropine-like amitriptyline, used to treat enuresis, may in susceptible patients produce paralytic ileus, as can phenothiazines when taken with anti-depressants or anti-Parkinsonian agents. Alcohol is a potent CNS depressant, as well as having varied effects on the gastro-intestinal tract; it slows the normal peristaltic action of the stomach. Studies show that gut function inhibition is potentiated by concurrent administration of amitriptyline and chlorpromazine, with the latter being more inhibitive. Constipation, blurred vision, and other atropine-like effects are common with imipramine and amitriptyline. Potentiation with alcohol can result in liver dysfunctions which may lead to death, and a patient on psychotropic drugs who is not warned against drinking alcohol, runs the risk of developing gastro-intestinal and other side effects. Most physicians acknowledge the dangers of polypharmacy, but these may occur unwittingly if the social habits of patients are ignored.

886. Milner, G., and Kakulas, B. A.
THE POTENTIATION BY AMITRIPTYLINE OF LIVER CHANGES INDUCED BY ETHANOL IN MICE.
 Pathology (Sydney), 1(2): 113-118 (10 ref.), 1969.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – liver, kidney
 – antidepressants – nutritive agents – *CAAAL-0 B-0388.

The effects of the interaction of amitriptyline and ethanol were evaluated in mice. The mice were given various combinations of amitriptyline, ethanol, and a placebo, each administered orally. Single doses of amitriptyline (50 mg/kg) or ethanol (25 ml/kg, 25% sol) induced slight to moderate fatty change of the liver; given together, the same dose of these preparations evoked severe fatty change. No significant changes were found in other organs. The possible implications, including the interaction of psychotropic drugs with ethanol as a cause of sudden death, are discussed.

887. Milner, G.
INTERACTION BETWEEN BARBITURATES, ALCOHOL AND SOME PSYCHOTROPIC DRUGS.
 Med. J. Aust. (Sydney), 1(24): 1204-1207 (37 ref.), 1970.
 E – general – DC (add., infra-add., unspec. incr.) – humans – CNS – antidepressants – barbiturates
 – sed., hypnot. – tranquilizers – *CAAAL-0 B-0538.

The author reviews the problem of interaction and experimentation reported elsewhere. With polypharmacy being practiced by many physicians and lay persons, serious side effects occur when one agent is taken in conjunction with another. Alcohol is more dangerous when potentiated with barbiturates than with tranquilizers, since as little as 0.5 mg/100 ml barbiturate with 0.1% alcohol has proved fatal. In a comparative study of barbiturate action, the group that took alcohol noted an increased severity of symptoms. In mice experiments, to compare potency of psychotropics with alcohol, it was found, using the time to regain the righting reflex as a measure of interaction potency, that chlorpromazine was the most potent, diazepam less potent, and thioridazine and trifluoperazine the least potent. Amitriptyline, trimipramine, and nortriptyline potentiated alcohol quite strongly, while in mice, desipramine and methylphenidate protected against an alcohol-induced coma. Barbiturates and alcohol still present the greatest danger of synergistic action, since 200 mg butabarbitalone can be potentiated by as little as half a litre of beer. Thus, non-barbiturate hypnotics such as diazepam and nitrazepam should be used in preference to barbiturates. There is a need for more education of physicians concerning these hazards, and for more careful prescription of barbiturates, the dangers of which sometimes far outweigh their usefulness.

888. Milner, G., and Landauer, A. A.
 ALCOHOL, THIORIDAZINE AND CHLORPROMAZINE EFFECTS ON SKILLS
 RELATED TO DRIVING BEHAVIOUR.
 Brit. J. Psychiat. (London), 118: 351-352 (6 ref.), 1971.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – acute admin. – in vivo
 – other drug lev. – mot. perform. – tranquilizers – *CAAAL-0 B-0961.

21 male students were randomly assigned to 3 equal groups, and received thioridazine, chlorpromazine, or placebo. A dose of 1.0 mg/kg was given at night, and a second 1.0 mg/kg dose was given in syrup the next morning. All subjects answered a questionnaire, and, 2 hr after the second drug dose, were given 3 motor skill tests: dot tracking, pursuit rotor, and simulated driving. After the tests, the subjects drank 0.8 ml/kg of diluted ethanol, resulting in a mean breathalyzer reading of 0.08%, and the tests were repeated. The results clearly showed that alcohol and the phenothiazines impaired motor skills. The phenothiazines tended to slow reaction times, chlorpromazine being the most potent. Both phenothiazines enhanced the sedative and motor skill-inhibiting effects of alcohol, chlorpromazine again exerting the stronger effect. Questionnaire results also indicated that chlorpromazine, and, to a lesser extent, thioridazine, added to the effects of alcohol intoxication.

889. Min, P.
 CHŌSEN-NINJIN NO JIKKENTEKI KENKYŪ. SONO ICHI: CHŌSEN-NINJIN O
 MOTTE SHIIKU SERU *RATTE* NI OKERU KIGASHIKEN OYOBİ NI SAN
 YAKUBUTSU NO CHŪDOKU-GENSHŌ NI OITE. [Experimental investigations of *Panax*
ginseng. I. Hunger experiments made with rats which were fed with *Panax ginseng*, and the
 influence of feeding with *Panax* on the toxicity of some drugs].
 Chosen Medical Association, Journal (Keijo), 19(1): 68-96 (59 ref.), 1929.
 CHŌSEN-NINJIN NO JIKKENTEKI KENKYŪ. SONO NI: CHŌSEN-NINJIN O MOTTE
 SHIIKU SERU *RATTE* NI OKERU NI SAN KEIREN-DOKU NO CHŪDOKU-GENSHŌ
 OYOBİ CHISHIRYŌ NI OITE. [Experimental investigations of *Panax ginseng*. II. The toxic
 effects of some convulsive poisons and the symptoms caused in rats fed *Panax ginseng*].
 Folia Pharmacol. Jap. (Nippon Yakurigaku Zasshi) (Kyoto), 9: 282-296 (14 ref.), 1930.
 CHŌSEN-NINJIN NO JIKKENTEKI KENKYŪ. SONO SAN: CHŌSEN-NINJIN O MOTTE
 SHIIKU SERU *RATTE* NI OKERU NI SAN MAHI-DOKU NO CHŪDOKU-GENSHŌ
 OYOBİ CHISHIRYŌ NI OITE. [Experimental investigations of *Panax ginseng*. III. The toxic
 effects of some paralyzing poisons and the symptoms caused in rats fed *Panax ginseng*].
 Folia Pharmacol. Jap. (Nippon Yakurigaku Zasshi) (Kyoto), 9: 310-324 (9 ref.), 1930.
 CHŌSEN-NINJIN NO JIKKENTEKI KENKYŪ SONO SHI: CHŌSEN-NINJIN O MOTTE
 SHIIKU SERU *RATTE* NI OKERU NI SAN SHINKEI-DOKU NO CHŪDOKU-GENSHŌ
 OYOBİ CHISHIRYŌ NI OITE. [Experimental investigations of *Panax ginseng*. IV. The toxicity
 of some nervous poisons and the poisonous effects caused by them in rats fed *Panax ginseng*].
 Folia Pharmacol. Jap. (Nippon Yakurigaku Zasshi) (Kyoto), 11: 238-255 (30 ref.), 1931.
 CHŌSEN-NINJIN NO JIKKENTEKI KENKYŪ. SONO GO: CHŌSEN-NINJIN TO
 BEIKOKU-NINJIN TO NO HIKAKU KENKYŪ. [Experimental investigations of *Panax*
ginseng. V. Comparative investigations of the chemical compounds and the general effects of
Panax ginseng and *Panax quinquefolius*].
 Folia Pharmacol. Jap. (Nippon Yakurigaku Zasshi) (Kyoto), 11: 256-260 (11 ref.), 1931.
 J – GS – exp. cont. – DC (decrease) – mammals – acute admin. – chronic admin. – in vivo – CNS
 – *CAAAL-0 A-0926.

In this series of experimental studies on *Panax ginseng*, the author investigates the effects of *Panax* on various drugs. The third communication shows the effects of *Panax* on the toxic symptoms and fatal dosage of alcohol, chloral hydrate, urethane, and veronal sodium. The min dosage of alcohol required to anesthetize rats fed *Panax ginseng* for 4 weeks was found to be 80 mg (100% alcohol ip), whereas it was 50.0 mg for controls not fed *Panax*. The fatal dosage for the former group was 120 mg (100% ip), and for the control group was 90 mg. It is concluded that the effect of alcohol on the

CNS is antagonized by the presence of *Panax ginseng*. Fewer alcoholized rats fed *Panax* fell asleep or died in comparison to other alcoholized rats.

890. Minot, A. S., and Cutler, J. T.
 GUANIDINE RETENTION AND CALCIUM RESERVE AS ANTAGONISTIC FACTORS
 IN CARBON TETRACHLORIDE AND CHLOROFORM POISONING.
 J. Clin. Invest. (Boston), 6: 369-402 (43 ref.), 1928-1929.
 E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo –
 CNS – anti-infectants – miscellaneous – *CAAAL-0 A-0927.

Carbon tetrachloride (CCl₄) poisoning was investigated in dogs. 5 dogs received 4 cc CCl₄ and 4 cc/kg alcohol. The increased toxicity of CCl₄-alcohol was manifested by much higher and more rapid rises in guanidine levels, with subsequent extreme hypoglycemia, soon followed by death. The increases in guanidine usually occurred even if meat was not eaten after the dose, the guanidine in such cases evidently being of endogenous origin. Alcohol alone caused no such accumulation of guanidine, even when large amounts of meat were eaten. Dogs on a high calcium meat diet usually failed to survive the combined dose unless given intensive calcium medication. If death is not caused by hemorrhages, which tend to be more severe after CCl₄-alcohol than when CCl₄ is given alone, this intoxication can be controlled by calcium therapy.

891. Minz, S., and Serianni, E.
 L'AZIONE DELL' ADRENALINA E DELL' ATROPINA SULL' ALCOOLEMIA
 PROVOCATA. [The action of adrenalin and atropine on induced alcoholemia].
 Accademia Nazionale dei Lincei, Rendiconti (Rome), ser. 6, 24: 235-238 (2 ref.), 1936.
 I – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in
 vivo – dose resp. – blood lev. – autonomic agents – cardiovasc. agents – *CAAAL-0 A-1424.

The effects on the blood alcohol curve (BAC) of sc injections of 1 cc adrenalin hydrochloride or 0.3-0.5 mg atropine sulphate, prior to ingestion of 0.5 cc/kg 95% alcohol in a 20% aqueous sol, were investigated in 6 healthy, fasting subjects. In some subjects, the tests were repeated several times. Each subject served as his own control, and blood alcohol concentrations were determined by the Widmark micromethod. In 2 subjects there was a significant deviation from the normal BAC, and, in these individuals, adrenalin and atropine had opposite effects. Adrenalin lowered the climax of the BAC by 25% in 1 of the cases, and by 20% in the other, while atropine increased the BAC climax by 20% and 5%, respectively. The most noticeable feature of the atropine effect was not so much the small increase in the climax, but rather the course of the BAC, which in all its phases manifested values clearly superior to the normal curve. In the other 4 subjects, the results were inconclusive.

892. Mirsky, I. A., and Nelson, N.
 THE INFLUENCE OF THE PANCREAS AND THE LIVER ON THE OXIDATION OF
 ETHYL ALCOHOL.
 Amer. J. Physiol. (Bethesda), 127: 308-314 (14 ref.), 1939.
 E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo –
 blood lev. – glands – liver, kidney – anesthetics – elect., water-bal. agents – hormones, hormone antag.
 – *CAAAL-236-A2 A-0928.

5 series of experiments were conducted on dogs and rabbits. In 1 experiment, the effect of chloroform anesthesia on alcohol utilization was investigated. The rate of removal of alcohol from the blood after an iv injection of 1 g/kg ethanol in 20% sol was determined in dogs under the following conditions: prior to chloroform anesthesia, after exposure to chloroform anesthesia for 15 min/day for 5 days, and 12 days after the cessation of chloroform anesthesia. It was found that chloroform anesthesia produces a definite depression of the rate at which alcohol is removed from the blood, which finding is in accord with the data that even a partial hepatectomy will decrease the utilization of alcohol.

893. Mirsky, I. A., Piker, P., Rosenbaum, M., and Lederer, H.
 "ADAPTATION" OF THE CENTRAL NERVOUS SYSTEM TO VARYING
 CONCENTRATIONS OF ALCOHOL IN THE BLOOD.
 Quart. J. Stud. Alcohol (New Haven), 2: 35-45 (17 ref.), 1941.
 E – exp. – DC (decrease) – drug-dep. humans – acute admin. – chronic admin. – in vivo – blood lev.
 – other drug lev. – CNS – stimulants – *CAAAL-134-A1 A-0929.

Experiments were conducted to test whether the blood alcohol concentration (BAC) is the only factor responsible for the development and maintenance of symptoms of intoxication. Preliminary tests on rabbits suggested that the CNS can adjust to a high alcohol concentration if enough time is permitted to elapse. Tests on 8 male alcoholics showed that, in all but 1 instance, patients became sober at a BAC higher than that at which they developed gross signs of intoxication. 2 subjects received sufficient amounts of ethanol po to induce coma, then 9 cc of a 10% metrazol sol was given by slow iv injection. Within 10 min, both subjects became conscious and responded to various verbal and painful stimuli. A blood sample taken from 1 subject immediately before the metrazol administration contained 437 mg% alcohol; 20 min later, the BAC was 438 mg%. The corresponding data for the second subject were 428 mg% BAC before metrazol, and 439 mg% BAC 11 min after metrazol. 6 hr after metrazol administration, no stigmata of alcoholic intoxication were observed in either subject; nevertheless, the BAC was 244 mg% in 1 subject, and 291 mg% in the other. The results suggest that a profound chemical stimulus of the CNS may restore function to some degree at a higher BAC than is otherwise possible.

894. Misra, P. S., Lefevre, A., Rubin, E., and Lieber, C. S.
 EFFECT OF ETHANOL INGESTION ON ETHANOL, MEPROBAMATE AND
 PENTOBARBITAL METABOLISM.
 Gastroenterology (Baltimore), 58(2): 308 (0 ref.), 1970.
 E – abst. – exp. cont. – cross-tol. – DC (decrease) – humans – mammals – acute admin. – chronic
 admin. – in vivo – in vitro – blood lev. – liver, kidney – metab. proc. – *CAAAL-0 B-0539.

The effect of ethanol on drugs known to be metabolized in liver microsomes was studied in rats. C¹⁴-meprobamate half-life was found to be 138±19 min in rats pretreated for 1 month with ethanol (36% of total calories), compared to 254±36 min in controls. In 5 human volunteers, the C¹⁴-meprobamate half-life was decreased by 50% after 4-6 weeks of alcohol administration (up to 46% of cal). After 1 g/kg ethanol po, the average blood ethanol clearance increased by 70%. The half-life of pentobarbital (6 mg/kg po) decreased by 20% in 3 volunteers after 3-4 weeks of ethanol. 1 month of ethanol feeding doubled the capacity of liver slices to convert meprobamate into polar metabolites in vitro. It is concluded that chronic ethanol administration accelerates the clearance of ethanol, meprobamate, and pentobarbital from the blood by stimulating the hepatic microsomal drug-metabolizing enzymes, such as the adaptive microsomal ethanol-oxidizing system, and the aniline, pentobarbital, and benzpyrene hydroxylases.

895. Mitchell, S. W.
 ON THE TREATMENT OF RATTLESNAKE BITES, WITH EXPERIMENTAL
 CRITICISMS UPON THE VARIOUS REMEDIES NOW IN USE.
 North American Medico-Chirurgical Review (Philadelphia), 5: 269-311 (24 ref.), 1861.
 E – exp. cont. – general – DC (unchanged) – mammals – acute admin. – in vivo – cardiovasc. – G.I.
 tract – respir. – respir. agents – *CAAAL-0 A-0930.

The author discusses various fallacies, general considerations, and proper research methods concerning snake-bite antidotes, the various symptoms and phenomena of rattlesnake bites, local treatment, and general or constitutional treatment. Experiments were conducted on pigeons and rabbits in which 2 or 3 drops of venom were mixed with various amounts of alcohol for various periods of time, or else venom was placed in a drachm of alcohol and the alcohol evaporated slowly; the dose was then

injected sc. In all cases, the subjects died. Experiments on dogs bitten by snakes or injected with venom, and then administered alcohol or whiskey po, showed that alcohol is not a direct antidote to venom, but can be considered as a useful, "stimulus to be employed to buoy the patient over the prostration caused by venom poisoning." The condition of stimulation, however, must be maintained. Profound intoxication is not a condition to be desired. Where oral administration is impractical, enemata of brandy and inhalations of hot alcohol may be used.

896. Moeschlin, S., and Garson, H.
 UNTERSUCHUNGEN ÜBER DIE ÄTHYLALKOHOLTHERAPIE DER
 EXPERIMENTELLEN METHYLALKOHOLVERGIFTUNG. [Investigations on the ethyl
 alcohol treatment of experimental methyl alcohol poisoning].
 Schweiz. Med. Wschr. (Basel), 85: 61-62 (21 ref.), 1955.
 G – ES – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – alcohols
 – *CAAAL-7267-N8 A-0931.

30 guinea pigs received 1 g/kg ethanol sc 15 min prior to 10 g/kg methanol ip. Ip injections of 0.4 g/kg ethanol were then given at regular intervals for 72 hr or until death. The percentages of survivors from groups of 30 ethanol-treated and 30 control animals, respectively, were: after 24 hr, 58% and 79%; after 48 hr, 32% and 37%; after 72 hr, 11% and 21%; and, after 96 hr, 5% and 5%. The results show that, if anything, ethanol speeded the death of methanol-poisoned animals. The ethanol dosages corresponded to those used by Røe in humans (Acta Med. Scand. (Stockholm), 126, Suppl. 182, 253 pp., 1946). It is concluded that the beneficial results obtained by Røe were due to the fact that ethanol treatment was combined with alkali administration. Early and intensive alkali treatment probably remains the most important measure in counteracting the toxic effects of methanol.

897. Mohr, L.
 UEBER BLUTVERÄNDERUNGEN BEI VERGIFTUNGEN MIT BENZOLKÖRPERN. [On
 blood changes by poisoning with benzol derivatives].
 Deutsch. Med. Wschr. (Stuttgart), 28(5): 73-76 (8 ref.), 1902.
 G – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – absorp., distrib., stor. –
 blood comp., sites, lymph – cardiovasc. – CNS – liver, kidney – indust. intox. – miscellaneous –
 *CAAAL-0 A-0932.

The author discusses case material regarding intoxications by exposure to aromatic substances (nitro- and chlorobenzene) complicated by alcoholic intake. The subjects, exposed to substituted benzene compounds, complained of headache, dizziness and difficulties in passing urine after alcohol ingestion. The author mentions the danger of alcohol to workers in aniline plants, and points out that alcohol and ether dissolve the nitrogroups in substituted benzene compounds, facilitating absorption in the organism. In this connection, degenerative and regenerative blood changes were observed resembling pernicious anemia. A number of references are made to other investigators who observed the same pathological changes, which were characterized by fatty degeneration of the liver, kidneys, and heart.

898. Molenda, R., and Obrzut, A.
 WPŁYW ETANOLU I FENAKTYLU NA CIEPŁOTĘ MIĘŚNI, MÓZGU, WĄTROBY I
 SKÓRY. [Effect of ethanol and fenactil on the temperature of muscle, brain, liver, and skin].
 Acta Physiol. Pol. (Warsaw), 16(5): 771-781 (16 ref.), 1965.
 Po – ES – RS – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo
 – dose resp. – cardiovasc. – CNS – liver, kidney – skel., muscle, skin – tranquilizers – *CAAAL-0
 B-0389.

41 rats received 0.25, 0.50, 1.25, or 2.50 g/kg ethanol; 1, 2.5, 5, or 10 mg/kg fenactil; or ethanol plus fenactil; and the temperatures of the muscle, brain, liver, and skin were determined with an electric

thermometer after a 20-min cooling of the immobilized rat, the ambient temperature averaging 23-25°C. Small doses of fenactil (2.5 mg/kg) and small doses of ethanol (0.5 g/kg) lowered the surface and intramuscular temperatures, but had little effect on the brain and liver temperatures. Higher doses of fenactil (5 mg/kg) and ethanol (1.25 g/kg) produced a marked drop in the surface, intramuscular, cerebral, and hepatic temperatures. Even small doses of fenactil in combination with ethanol resulted, due to synergistic action, in a pronounced temperature fall, not only on the skin surface and in the muscles, but also in the internal organs, brain, and liver.

899. Molenda, R., and Obrzut, A.

EFFECT OF ETHANOL AND PHENACTIL ON THE TEMPERATURE OF MUSCLE, BRAIN, LIVER AND SKIN.

Acta Physiol. Pol. (English Translation) (Warsaw), 16(5): 657-665 (16 ref.), 1965.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp.
– cardiovasc. – CNS – liver, kidney – skel., muscle, skin – tranquilizers – *CAAAL-0 B-0390.

41 rats received 0.25, 0.50, 1.25, or 2.50 g/kg ethanol; 1, 2.5, 5, or 10 mg/kg fenactil; or ethanol plus fenactil; and the temperatures of the muscle, brain, liver, and skin were determined with an electric thermometer after a 20-min cooling of the immobilized rat, the ambient temperature averaging 23-25°C. Small doses of fenactil (2.5 mg/kg) and small doses of ethanol (0.5 g/kg) lowered the surface and intramuscular temperatures, but had little effect on the brain and liver temperatures. Higher doses of fenactil (5 mg/kg) and ethanol (1.25 g/kg) produced a marked drop in the surface, intramuscular, cerebral, and hepatic temperatures. Even small doses of fenactil in combination with ethanol resulted, due to synergistic action, in a pronounced temperature fall, not only on the skin surface and in the muscles, but also in the internal organs, brain, and liver.

900. Molenda, R.

BADANIA NAD SKOJARZONYM DZIAŁANIEM CHLOROPROMAZYNY I ETANOLU.

[Investigations on the combined effects of chlorpromazine and ethanol].

Roczn. Akad. Med. Marchlewski (Białystok), 12: 93-107 (73 ref.), 1966.
Po – ES – RS – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo
– dose resp. – CNS – metab. proc. – tranquilizers – *CAAAL-0 B-0576.

The purpose of the study was to determine whether the effect of ethanol and chlorpromazine is synergistic, and, if so, what type of synergism it is, and whether this synergism is connected with disorders in the elimination of ethanol. First, the LD₅₀ of ethanol and chlorpromazine were determined in white mice using the method of Lichtfield and Wilcoxon. The results were tabulated and evaluated on the Gaddum coordinate system. The changes in the conditioned reflexes were studied in hooded rats weighing 200-400 g, using 0.2% chlorpromazine given ip and 35% alcohol given into the stomach. The controls received a physiological saline sol. The elimination of alcohol was studied in rats in 2 groups—using 20 mg/kg of 2.5% chlorpromazine and 1.4 g/kg of 25% alcohol, in one group, and, in the second group, 100 mg/kg of chlorpromazine and 1.8 g/kg of ethanol. The results were tabulated and graphically evaluated. The author concludes that the effect of ethanol and chlorpromazine is a synergism of the additive type, especially when both ethanol and chlorpromazine are given in small doses. Small doses of chlorpromazine increase the blood alcohol level, but only large doses prolong the time of elimination of ethanol.

901. Molenda, R., and Obrzut, A.

DZIAŁANIE CHLOROPROMAZYNY, ETANOLU I REZERPINY NA CIEPŁOTĘ NARZĄDOWĄ PRZY PORĄŻENIU UKŁADU SYMPATYCZNEGO I PARASYMPATYCZNEGO. [Effect of chlorpromazine, ethanol and reserpine on organ temperature with sympathetic and parasympathetic block].

Przegl. Lek. (Cracow), 22(8): 544-547 (18 ref.), 1966.

Po – ES – RS – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – liver, kidney – skel., muscle, skin – autonomic agents – *CAAAL-12258-A1 B-0391.

A total of 130 experimental and 10 control rats of both sexes were immobilized for 20 min at 22 to 23°C. Dihydroergotamine (im) and atropine (im) were used to block the sympathetic and parasympathetic system. Chlorpromazine (ip), 1.25 g/kg ethanol as a 32% sol (ip), and reserpine (im) were administered, and the liver and thigh muscle temperatures recorded with an electric thermometer. After 80 min, the liver temperature of the controls dropped from 37.9 to 37.3°C; after ethanol, it dropped from 38.2 to 36.2°C; after ethanol plus 1 mg/kg dihydroergotamine, it dropped from 38.3 to 36.3°C; and after ethanol plus 2 mg/kg atropine, it dropped from 37.9 to 36.3°C. After 80 min, the muscle temperature of the controls dropped from 36.7 to 36.3°C; after ethanol, it dropped from 37.1 to 34.6°C; after ethanol plus 1 mg/kg dihydroergotamine it dropped from 37.3 to 35.2°C; and after ethanol plus 2 mg/kg atropine it dropped from 36.9 to 35.1°C. It is concluded that dihydroergotamine and atropine do not increase the hypothermic action of ethanol in muscle and liver.

902. Molenda, R.

OCENA SKOJARZONEGO DZIAŁANIA ALKOHOLU ETYLOWEGO I HYDRAZYDU KWASU IZONIKOTYNOWEGO PRZY POMOCY BADANIA ODRUCHÓW WARUNKOWYCH. [Estimation of the combined effect of ethanol and isonicotinic acid hydrazide by the method of conditioned reflexes].

Przegl. Lek. (Cracow), 23(4): 399-403 (17 ref.),

1967.

Po – ES – RS – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – mot. perform. – CNS – anti-infectants – *CAAAL-0 B-0392.

Controlled experiments were carried out on the combined effect of ethanol and isonicotinic acid hydrazide (INH) at room temperature on 20 rats with trained avoidance reflexes conditioned to sound (10 rats) and light (10 rats). The criteria of evaluation were the degree of reduction in conditioned avoidance reflexes and the change in reaction time. Alcohol (35%) was introduced po in doses of 3 g/kg, and, simultaneously, INH in 10% aqueous sol was given im in a dose of 100 mg/kg. It was found that INH prolonged and potentiated the neuroplegic action of alcohol, with regard to avoidance reflexes in rats conditioned to sound and light. The prolonged reaction time evoked by alcohol in rats conditioned to sound was not potentiated by INH, but INH clearly prolonged the reaction time in rats conditioned to light.

903. Molenda, R.

OCENA SKOJARZONEGO DZIAŁANIA CHLOROPROMAZYNY I ETANOLU NA PODSTAWIE BADANIA ODRUCHÓW WARUNKOWYCH. [Evaluation of the combined effect of chlorpromazine and ethanol on avoidance reflex in rats].

Przegl. Lek. (Cracow), 24(5): 493-495 (21 ref.),

1968.

Po – ES – RS – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – psychol. perform. – CNS – tranquilizers – *CAAAL-0 B-0393.

Studies of the combined effect of chlorpromazine and ethanol on avoidance reflexes in rats were performed. It was found that avoidance reflex was partially inhibited and reaction time was prolonged after individual administration of chlorpromazine (2.5 mg/kg) and ethanol (35%, 3 g/kg). Chlorpromazine increased the effect of ethanol; the combined effect was a simple addition of the individual effects.

904. Molenda, R.

ZACHOWANIE SIĘ OSTREJ TOKSYCZNOŚCI ETANOLU W OBECNOŚCI CHLOROPLOMAZYNY [S/C]. [Acute toxicity of ethanol given with chlorpromazine].

Przegl. Lek. (Cracow), 24: 611-613 (19 ref.),

1968.

Po – ES – RS – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – metab. proc. – respir. – tranquilizers – *CAAAL-0 B-0577.

The object of the experiment was to determine whether chlorpromazine increases the toxic effects of alcohol after simultaneous administration. First, the LD₅₀ was determined for ethanol and chlorpromazine in white mice (weighing about 20 g), using the method of Lichtfield and Wilcoxon. The results were evaluated by the Gaddum coordinate system. The LD₅₀'s of the chlorpromazine and alcohol were found to be 200 mg/kg and 4700 mg/kg, respectively. After administration of high alcohol or chlorpromazine doses, convulsions leading to rapid death were observed. Most animals showed an inhibition of metabolism, as a result of which, disorders in the respiratory centre and a decrease of body temperature were observed. The author concludes that the synergistic effect of ethanol and chlorpromazine is additive. This effect is the strongest after administration of large toxic doses of ethanol with small toxic doses of chlorpromazine, and is smallest when large toxic doses of ethanol are combined with large toxic doses of chlorpromazine.

905. Molenda, R.

WPŁYW CHLOROPROMAZYNY NA ELIMINACJĘ ALKOHOLU. [The effect of chlorpromazine on alcohol elimination].

Przegl. Lek. (Cracow), 24: 663-665 (20 ref.),

1968.

Po – ES – RS – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – CNS – cardiovasc. – absorp., distrib., stor. – tranquilizers – *CAAAL-0 B-0575.

The increased effect of alcohol after chlorpromazine administration is considered to be due to a slowdown in the elimination of alcohol. Thus, it was investigated whether small doses of chlorpromazine prolong the time of elimination of alcohol, or if they only induce changes in the process of elimination. The experiments were performed on 2 groups of rats—the first group of 10 rats received 20 mg/kg 2.5% chlorpromazine and 1400 mg/kg 25% alcohol; 10 control rats received a physiological saline sol and the above amount of alcohol. The second group of 12 rats received 100 mg/kg chlorpromazine, and, after 2 hr, 1800 mg/kg of alcohol; control rats received saline and alcohol. The blood test results were tabulated, and curves of elimination were plotted. Small doses of chlorpromazine were found to increase the blood alcohol only briefly, and did not prolong the time of elimination. Toxic doses, however, both increased the blood alcohol concentration and prolonged the time of elimination. The author concludes that the irregular course of the alcohol elimination curve, when alcohol is administered simultaneously with chlorpromazine, could also be due to hemodynamic disturbances, as well as to change of permeability of blood vessels.

906. Molinari, G.

ALCOOLTERAPIA ENDOVENOSA NEL REUMATISMO CARDIACO EVOLUTIVO E NELLE INTOSSICAZIONI DA BARBITURICI. [Intravenous alcohol therapy in progressive cardiac rheumatism and barbituric intoxication].

Riforma Medica (Naples), 50: 1732 and 1735 (3 ref.),

1934.

I – review – DC (antidotal) – humans – drug-dep. humans – barbiturates – elect., water-bal. agents – *CAAAL-0 A-0933.

A review of literature is given on the therapeutic effect of iv injections of alcohol in cardiac disease and barbiturate intoxication. The treatment in cardiovascular disease is initiated with 5 cc iv alcohol in a 20% glucose serum, followed by 8-10 cc 33% alcohol sol in an isotonic glucose serum. According to the authors cited, the results are most encouraging. In previously reported cases of intoxication by gardenal (1.50-2 g), iv injections of 30% alcohol were given (4 injections, 20 cc each—60 cc during 6 hr, followed by another 20 cc the following day). The alcohol served as an effective antidote in restoring stability to the patient by the third day.

907. Møller, K. O.
 DØD FREMKALDT MED TERAPEUTISKE DOSER AF MORFIN ELLER
 MORFIN-SKOPOLAMIN HOS ALKOHOLPÅVIRKEDE ELLER BARBITURSYRE
 PÅVIRKEDE PERSONER. [Death caused by therapeutic doses of morphine or
 morphine-scopolamine in persons under the influence of alcohol or barbiturates].
 Ugeskr. Laeg. (Copenhagen), 114(50): 1785-1793 (8 ref.), 1952.
 Da – general – DC (add., infra-add., unspec. incr.) – med.-leg. – post.-mort. – humans – drug-dep.
 humans – blood lev. – other drug lev. – respir. – analg., antipyret. – autonomic agents – barbiturates
 – *CAAAL-6382-D3 A-0934.

17 cases of fatal poisoning due to the synergism of alcohol and barbiturates with morphine and morphine-scopolamine are reported, with individual commentaries for each case. In all 7 cases concerning alcohol combinations, there was a high blood alcohol concentration when the drugs were administered—about 1.8°/oo in 1 case, and between 2.2 and 2.7°/oo in the others. In 3 cases, the injection of 0.3-0.4 mg/kg morphine caused death in persons with a blood alcohol level of 2.2-2.5°/oo; the author calculates that in these cases the blood alcohol reached about 62-70% of the minimum lethal concentration, and the morphine reached about 20-25% of the minimum lethal dose. The evidence does not permit a decision as to whether the synergism is additive or potentiative.

908. Møller, K. O.
 DØD FREMKALDT MED TERAPEUTISKE DOSER AF MORFIN ELLER
 MORFIN-SKOPOLAMIN HOS ALKOHOLPÅVIRKEDE ELLER BARBITURSYRE
 PÅVIRKEDE PERSONER. [Death from therapeutic doses of morphine or
 morphine-scopolamine in persons affected by alcohol or barbituric acid].
 T. Norsk. Laegeforen. (Oslo), 73(1): 1-8 (8 ref.), 1953.
 N – general – DC (add., infra-add., unspec. incr.) – med.-leg. – post.-mort. – humans – drug-dep.
 humans – blood lev. – other drug lev. – respir. – analg., antipyret. – autonomic agents – barbiturates
 – *CAAAL-6382-D3 A-0935.

17 cases of fatal poisoning due to the synergism of alcohol and barbiturates with morphine and morphine-scopolamine are reported, with individual commentaries for each case. In all 7 cases concerning alcohol combinations, there was a high blood alcohol concentration when the drugs were administered—about 1.8°/oo in 1 case, and between 2.2 and 2.7°/oo in the others. In 3 cases, the injection of 0.3-0.4 mg/kg morphine caused death in persons with a blood alcohol level of 2.2-2.5°/oo; the author calculates that in these cases the blood alcohol reached about 62-70% of the minimum lethal concentration, and the morphine reached about 20-25% of the minimum lethal dose. The evidence does not permit a decision as to whether the synergism is additive or potentiative.

909. Møller, K. O.
 DEATH FROM THERAPEUTIC DOSES OF MORPHINE OR
 MORPHINE-SCOPOLAMINE IN PERSONS AFFECTED BY ALCOHOL OR
 BARBITURIC ACID.
 Bulletin on Narcotics (Lake Success, N.Y.), 5(4): 11-19 (8 ref.), 1953.
 E – general – DC (add., infra-add., unspec. incr.) – med.-leg. – post.-mort. – humans – drug-dep.
 humans – blood lev. – other drug lev. – respir. – autonomic agents – barbiturates – *CAAAL-6382-D3
 A-0936.

17 cases of fatal poisoning due to the synergism of alcohol and barbiturates with morphine and morphine-scopolamine are reported, with individual commentaries for each case. In all 7 cases concerning alcohol combinations, there was a high blood alcohol concentration when the drugs were administered—about 1.8°/oo in 1 case, and between 2.2 and 2.7°/oo in the others. In 3 cases, the injection of 0.3-0.4 mg/kg morphine caused death in persons with a blood alcohol level of 2.2-2.5°/oo; the author calculates that in these cases the blood alcohol reached about 62-70% of the

minimum lethal concentration, and the morphine reached about 20-25% of the minimum lethal dose. The evidence does not permit a decision as to whether the synergism is additive or potentiative.

910. Møller, K. O.

BARBITURATES AND ALCOHOL.

Lancet (London), 264: 1253 (1 ref.),

1953.

E – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – analg., antipyret. – autonomic agents – barbiturates – *CAAAL-0 A-0937.

The author refers to clinical data and chemical analyses which show that the intake of relatively moderate doses of barbiturates (especially iv) plus relatively large doses of alcohol, and of alcohol by persons with chronic bromism, may prove fatal. The injection of therapeutic doses of morphine salts (15-30 mg) or morphine-scopolamine in persons with 0.18-0.2% or more of alcohol in the blood can also effect an often fatal poisoning, with all the symptoms of morphine poisoning.

911. Montale, P., Peris, G., Marchese, S., and Tedoldi, A.

LA REAZIONE VASOMOTORIA ALL'ALCOOL IN CORSO DI TRATTAMENTO CON FURALTADONE: OSSERVAZIONI CARDIO-ANGIOLOGICHE. [The vasomotor reaction to alcohol during treatment with furaltadone: cardioangiological observations].

Arch. Maragliano Pat. Clin. (Genoa), 18: 625-639 (31 ref.),

1962.

I – exp. – DC (sensit.) – humans – acute admin. – chronic admin. – in vivo – cardiovasc. – metab. proc. – *CAAAL-10683-B1 A-1425.

An experimental study was conducted on 2 groups of patients undergoing treatment with furaltadone, to clarify the cardiovascular reaction following the combined intake of the drug with alcohol. The first group of 10 subjects (16-45 yr) was pretreated with 1 g/day (4 x 250 mg) furaltadone for 5 days. On the fifth day, 50 cc cognac was given, and subjective and objective reactions, and EEG, distal oscillograph, and digital plethysmograph recordings were made. A second group of 30 patients (19-55 yr) underwent the same treatment, except that wine and beer were substituted for cognac, and no instrumental recordings were made. 60-70% of the subjects showed alcohol intolerance symptoms, such as dizziness, nausea, shivering, general malaise, heat sensations, etc., which were similar to the antabuse-alcohol reaction, and which varied in extent and intensity. It is concluded that furaltadone interferes with the metabolic degradation of alcohol, and abstinence during treatment with the drug is advised.

912. Montañés del Olmo, E.

ÓXIDO DE CARBONO Y ALCOHOLISMO AGUDO. [Carbon monoxide and acute alcoholism].

Clin. Lab. (Zaragoza), 34: 442-444 (0 ref.),

1942.

Sp – general – DC (add., infra-add., unspec. incr.) – post-mort. – humans – blood comp., sites, lymph – cardiovasc. – metab. proc. – respir. – indust. intox. – *CAAAL-4774-E7 A-0938.

2 fatal cases are reported of alcohol intoxication followed by carbon monoxide (CO) poisoning in poorly-ventilated rooms. It was found that the alcohol contributed to the deaths, and increased the severity of the CO intoxication. The tachycardia and tachypnea which occur in the first periods of acute alcoholism bring about a more rapid fixation of CO by hemoglobin, by increasing the ventilation of the pulmonary gaseous exchange; the increase of organic combustion is accompanied by greater expenditure of oxygen, so that, concomitantly with CO intoxication, the blood is unable to provide enough oxygen to the tissues. All increases in oxygen expenditure aggravate the symptoms of intoxication.

913. Montoya, G., Bardisa, L., and Merino, A.
EFFECTO ANALGÉSICO DEL ETANOL Y SUS MODIFICACIONES POR RESERPINA, DOPAMINA Y NIALAMIDA. [Analgesic effect of ethanol, and its modifications by reserpine, dopamine, and nialamide].
 Archivos de Biología y Medicina Experimentales (Santiago), 6(1-3): R 23 (0 ref.), 1969.
 Sp – abst. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo
 – dose resp. – CNS – antidepressants – sed., hypnot. – tranquilizers – *CAAAL-0 B-1016.

The analgesic effect of ethanol, and the modifications of this effect by reserpine, nialamide, and dopamine, were studied in rabbits. Pain was induced by electrical stimulation of the dental pulp. Ethanol (0.6, 0.75, and 1.05 g/kg) was given alone and after administration of 1 of the other above-mentioned drugs. The ethanol successfully elevated the pain threshold of the animals. Reserpine, given 4 hr prior to the ethanol, antagonized the analgesic effect, whereas nialamide, given 6 hr before the ethanol, enhanced the effect. Dopamine had no modifying action on the ethanol effect.

914. Moon, H. D.
PATHOLOGY OF ACUTE CARBON TETRACHLORIDE TOXICITY.
 Amer. J. Path. (New York), 25: 788-789 (0 ref.), 1949.
 E – abst. – stat. surv. – DC (add., infra-add., unspec. incr.) – drug-dep. humans – liver, kidney – respir.
 – anti-infectants – *CAAAL-5197-E3 A-0939.

Of 12 fatal cases of carbon tetrachloride poisoning, 11 were associated with acute and chronic alcoholism. Death followed poisoning by 4 to 18 days, thus allowing determination of changes at various intervals after poisoning. Central lobular necrosis in the liver, renal tubular degeneration, and pulmonary edema were consistently present in all cases. A relationship is felt to exist between the rapidity of poisoning and the degree of alcoholism. The full text is published in Amer. J. Path. (New York), 26: 1041-1057, 1950.

915. Moon, H. D.
THE PATHOLOGY OF FATAL CARBON TETRACHLORIDE POISONING WITH SPECIAL REFERENCE TO THE HISTOGENESIS OF THE HEPATIC AND RENAL LESIONS.
 Amer. J. Path. (New York), 26: 1041-1057 (15 ref.), 1950.
 E – stat. surv. – DC (add., infra-add., unspec. incr.) – post-mort. – drug-dep. humans – acid-base, blood pH, elect. – blood comp., sites, lymph – cardiovasc. – G.I. tract – glands – liver, kidney – respir.
 – senses – anti-infectants – *CAAAL-0 A-0940.

Pathological changes in 12 cases of fatal poisoning due to carbon tetrachloride (CCl₄) are presented. The interval between exposure and death varied from 4-18 days, thus allowing determination of changes at various intervals after poisoning. Of the 12 cases, 11 were associated with acute or chronic alcoholism. 7 persons ingested CCl₄ accidentally or with suicidal intent, and 5 inhaled fumes; there was no difference observed in the lesions resulting from these 2 types of exposure. Hepatic lesions in persons dying soon after exposure were very extensive; the severity of the condition diminished with longer periods of survival. Renal lesions in persons dying within a few days after exposure were relatively slight. Renal morphological changes became progressively more pronounced with longer survival periods. Pulmonary edema was consistently present, and was greater in those who survived for longer periods. The author concludes that the frequent occurrence of a history of alcoholism in cases of fatal CCl₄ poisoning indicates a synergistic nephrotoxic, as well as hepatotoxic, effect between alcohol and CCl₄.

916. Moore, M., Raymond, A. F., and Gray, M. G.
ALCOHOLISM AND THE USE OF DRUGS: A REVIEW OF 841 CASES DIAGNOSED "WITH PSYCHOSIS DUE TO DRUGS AND OTHER EXOGENOUS TOXINS" OR

"WITHOUT PSYCHOSIS: DRUG ADDICTION".

Quart. J. Stud. Alcohol (New Haven), 2: 496-504 (12 ref.), 1941.
 E – stat. surv. – conj. addict. – psychot. humans – drug-dep. humans – psychol. perform. – species
 or sex diff. – barbiturates – *CAAAL-0 A-0941.

The association between the use of drugs and the intemperate use of alcohol is pointed out. Among first admissions with psychosis associated with the use of drugs, 50.4% of males and 25.5% of females were intemperate ("intemperance" was inferred from: repeated intoxication; physical, mental, or moral deterioration, or any disease due to alcohol; and unsocial acts due to alcohol). Of all admissions, 32.0% of males and 6.0% of females with psychoses were intemperate. 74.3% of readmitted male patients were intemperate. It is suggested that the intemperate use of alcohol is an important contributory factor in causing the readmission of patients with recurrences of drug psychoses.

917. Moragne, N. H.

BITE OF A COPPERHEAD—"TRIGONOCEPHALUS CONTORTIX"—TREATED WITH WHISKEY.

Southern Medical and Surgical Journal (Augusta), nsv 9(2): 81-82 (0 ref.), 1853.
 E – general – case hist. – DC (antidotal) – humans – *CAAAL-0 A-0942.

A case of poisonous snake bite treated with alcohol is described. A man was bitten near the ankle by a *Trigonocephalus contortix*, more commonly known as a copperhead. When the author was called in to treat him, he found the patient partially delirious, skin hot and dry, pulse 100 to 120, and the leg greatly swollen. A ligature was applied above the wound, and the patient was given whiskey ad libitum. By the 3rd day, the delirium had passed; the patient began to speak rationally and was on his way to recovery. By way of comparison, a second case is cited in which no alcohol was administered, and the patient died.

918. Morey, H. C.

A STRYCHNIA EATER—STRYCHNIA AN ANTIDOTE TO ALCOHOL.

Pacific Medical and Surgical Journal (San Francisco), 16(4): 540-542 (0 ref.), 1875.
 E – general – case hist. – DC (antidotal) – humans – CNS – stimulants – *CAAAL-0 A-0943.

In reply to a request from the editors of the journal, a request which was prompted by newspaper reports, the author describes his personal experience with a man who frequently drank, and had, for nearly 20 yr, used strychnine to sober up, the quantity depending upon the amount of alcohol consumed. The strychnine calmed his nervous condition, and completely restored him. "After a two weeks' drunk, with all the appearances of approaching delirium tremens, he got up in the morning with his mind clear, his eyes bright, his skin clear and fair, and with all the appearances of a man in perfect health and vigor, and ate as hearty a breakfast as usual, and went to his work as though he had never taken a drop of whiskey in his life." The author concludes from his own experimentation that strychnine is a beneficial antidote to the effects of alcohol.

919. Morgan, A. F., Brinner, L., Plaa, C. B., and Stone, M. M.

UTILIZATION OF CALORIES FROM ALCOHOL AND WINES AND THEIR EFFECTS ON CHOLESTEROL METABOLISM.

Amer. J. Physiol. (Bethesda), 189: 290-296 (25 ref.), 1957.
 E – SEC – exp. cont. – congen. stud. – mammals – chronic admin. – in vivo – blood lev. – species
 or sex diff. – liver, kidney – metab. proc. – *CAAAL-8300-D2 A-1426.

The utilization of calories for growth was investigated in young rats given an adequate diet, free access to drinking water, and supplements of 15 or 20% alcohol sol, wines (red, rosé, or white) of the same alcohol concentration, or water. Similar experiments were conducted on hamsters. It was found that

utilization of calories by rats given alcohol or wine was equal to controls given water. The growth of rats given red wine lagged behind that of the other groups, possibly due to digestive losses caused by the higher content of tannins and tartrates in this wine. When no additional water was given with the alcohol, or when water intake was restricted to the amount taken by the alcohol groups, food intake and growth decreased. When 1% cholesterol was added, all rats grew better than on the basal diet, and water restriction had less effect. Both liver and adrenal cholesterol were much increased by the exogenous cholesterol in all groups, but least in the wine-fed animals. Hamsters were little affected by water restriction, but growth was decreased on the cholesterol diet. Serum cholesterol was much increased, but least in the wine-fed groups.

920. Morgan, E. L., Wyatt, J. P., and Sutherland, R. B.
 AN EPISODE OF CARBON TETRACHLORIDE POISONING WITH RENAL
 COMPLICATIONS.
 Canad. Med. Ass. J. (Toronto), 60: 145-150 (0 ref.), 1949.
 E – SEC – general – DC (add., infra-add., unspec. incr.) – post-mort. – humans – blood comp., sites,
 lymph – cardiovasc. – CNS – G.I. tract – liver, kidney – respir. – anti-infectants – *CAAAL-0
 A-0944.

A report is given of 10 men who were exposed to carbon tetrachloride (CCl_4) fumes in a poorly-ventilated area for several hr. The CCl_4 concentration in the air was later estimated at about 10,000 parts/million. 3 men became ill and required medical attention, and 1 died 9 days after exposure. The CCl_4 exposure occurred just after Christmas celebrations, and, of the 3 cases of poisoning, 1 admitted having consumed alcohol, and the man who died was a fairly regular drinker of beer. The author notes that, "though one cannot conclude that consumption of alcohol enhances the toxicity of the chlorinated hydrocarbon, the frequency with which a history of ingestion of alcohol has been noted in cases of serious carbon tetrachloride poisoning causes one to wonder if this fact may not be significant."

921. Morgan, J. C., and Di Luzio, N. R.
 INHIBITION OF THE ACUTE ETHANOL-INDUCED FATTY LIVER BY PYRAZOLE.
 Proc. Soc. Exp. Biol. Med. (New York), 134(2): 462-466 (23 ref.), 1970.
 E – exp. comp. – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in
 vivo – blood lev. – mot. perform. – metab. proc. – *CAAAL-0 B-0578.

In order to separate toxic effects of ethanol *per se* from those resulting from ethanol metabolites, the influence of pyrazole (an inhibitor of ethanol metabolism) administration on the induction of the acute ethanol-induced fatty liver was studied. 36 mg pyrazole/100 g was administered ip to rats, followed 4 hr later by 6 g ethanol/kg po. A control group received only ethanol. 20 or 44 hr later, the rats were killed, and liver and plasma triglycerides determined. In the control group, the blood alcohol level (BAL) had decreased 88% from its 3 hr level after 20 hr; in the pyrazole-treated group, it was unaltered. The liver triglyceride concentration in the pyrazole group was also unaltered 20 hr later, whereas it had increased 5 times in the control group. 44 hr later, both groups had normal liver triglyceride concentration; the BAL was still elevated in the pyrazole group, however. It is concluded that the accumulation of triglyceride in liver is related to ethanol metabolites, rather than to ethanol itself. The failure of fatty liver to occur in the pyrazole-treated rats rules out a contributing role of ethanol intoxication, and its nervous system manifestations, on fatty liver development.

922. Morin
 EMPOISONNEMENT PAR LA STRYCHNINE GUÉRI À L'AIDE DE L'ALCOOL PUR.
 [Strychnine poisoning cured with the aid of pure alcohol].
 Annales de Thérapeutique Médicale et Chirurgicale et de Toxicologie (Paris), 2: 112 (0 ref.), 1844.
 F – general – case hist. – DC (antidotal) – humans – cardiovasc. – CNS – skel., muscle, skin –
 stimulants – *CAAAL-0 A-0945.

In a letter to the editor, the author describes treatment of a patient admitted to the hospital because of hemiplegia. The patient was given strychnine after other remedies failed. For the first 3 days, 2 1/2 cg/day were administered, and, on the fourth day, the dosage was raised to 5 cg/day in 6 tablets. On the fourth day, 1 hr after taking the 4th tablet, the patient began to feel sick. She trembled lightly, then the limbs stiffened as in tetanus, and the body was supported only by the head and heels. All the muscles were extremely contracted, the skin cold and pale, heart-beat imperceptible, and speech incoherent. The physician concluded that the patient was suffering from strychnine poisoning, and administered 30 g (1 oz) 36% alcohol by injection. Almost immediately, the patient's condition returned to normal.

923. Morselli, P. L., Veneroni, E., Zaccala, M., and Bizzi, A.
FURTHER OBSERVATIONS ON THE INTERACTION BETWEEN ETHANOL AND PSYCHOTROPIC DRUGS.
 Arzneimittelforschung (Aulendorf), 21(1): 20-23 (23 ref.), 1971.
 E – GS – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – chronic admin.
 – in vivo – blood lev. – mot. perform. – CNS – barbiturates – tranquilizers – *CAAAL-0
 B-0962.

14 female humans received 0.25 g/kg ethanol (equal to 2-3 glasses of wine) in aqueous sol po daily for 20 days. Blood samples were collected 15, 30, 60, and 180 min after administration. At the end of this period, the subjects were randomly divided into 4 groups, and treated daily with normal maintenance therapeutic drug doses for 10 days. Group A received 200 mg chlorpromazine/day, group B 15 mg diazepam/day, group C 200 mg phenobarbital/day, and group D 6 mg haloperidol/day. On the eleventh day, groups A,B,C, and D received 50 mg chlorpromazine, 5 mg diazepam, 50 mg phenobarbital, and 2 mg haloperidol, respectively, 30 min before the administration of 0.25 g/kg ethanol po, and blood samples were collected as before. Each subject acted as her own control. In both sessions, during the first 3 hr after alcohol ingestion, all subjects performed an easy embroidery task. All 4 drugs potentiated the effects of alcohol, although only haloperidol increased blood ethanol levels. Impairment was minimal for diazepam and chlorpromazine, but was very intense for phenobarbital and haloperidol, with marked side effects.

924. Mueller, B.
BEHAUPTETE UND WIRKLICHE FEHLERQUELLEN BEI DER BLUTALKOHOLBESTIMMUNG. [Claimed and real errors in blood alcohol tests].
 Kriminalistik (Berlin), 12: 81-84 (7 ref.), 1938.
 G – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – med.-leg.
 – mot. vehic. – humans – blood lev. – psychol. perform. – analg., antipyret. – barbiturates – hormones,
 hormone antag. – stimulants – *CAAAL-0
 A-0946.

In a general discussion, it is pointed out that nicotine in combination with alcohol potentiates the psychic intoxication phenomena without affecting the blood alcohol level, whereas insulin decreases the blood alcohol level. Tests revealed that insulin is not an effective sobering agent, even when administered simultaneously with alcohol. Aspirin has no effect on blood alcohol. Case material is noted involving intake of 4 veronal tablets and alcohol, followed by falling asleep while in control of a motor vehicle. The author concludes that there are a number of factors to be considered when evaluating the blood alcohol level, in order to pass judgement in traffic offences. Concerted effort on the part of various authorities is called for in automotive medicine.

925. Mueller, B.
DIE BEWERTUNG VON BLUTALKOHOLBEFUNDEN. [The assessment of blood alcohol findings].
 Munchen. Med. Wschr. (Munich), 92: 127-134 (38 ref.), 1950.

G – SEC – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev. – other drug lev. – mot. perform. – psychol. perform. – absorp., distrib., stor. – CNS – analg., antipyret. – barbiturates – hormones, hormone antag. – nutritive agents – sed., hypnot. – *CAAAL-5472-A1 A-1454.

The evaluation of blood alcohol tests is discussed, with special reference to the validity of, and possible sources of error in, present methods of determination, as well as the applicability of legal blood alcohol levels (BAL), and conditions affecting the state of intoxication and the BAL. Evaluations of the degree of intoxication cannot be invariably precise, due to the possible action of agents, such as acetone, which alter the test results and can give a false positive reading of up to 0.85‰. Among substances which affect the state of intoxication are hypnotics, aspirin, and pyramidon, which increase the alcohol effect, especially fatigue. Veramon, in combination with a small quantity of alcohol, can produce intoxication symptoms. On the other hand, caffeine and pervitin improve performance after alcohol ingestion, although the BAL is not affected. Insulin and cane sugar reduce both the BAL and the psychic effects of alcohol. The author disagrees with the opinion that sweetened alcoholic beverages have a stronger intoxicating effect than unsweetened drinks.

926. Mueller, B.
FRAGEKASTEN: FRAGE 110. [Correspondence: Question 110].
Munchen. Med. Wschr. (Munich), 103: 2242-2243 (0 ref.), 1961.
G – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – liver, kidney – analg., antipyret. – barbiturates – tranquilizers – *CAAAL-0 A-0947.

In a query to the editor, 3 questions are asked. To the question, "Do drugs, like luminal, codeine, meprobamate, and dolantin have a cumulative effect with alcohol?" the answer is given that the blood alcohol level is not increased, but the alcohol effect is increased. The answer to the second question, "Is the oxidation, owing to a liver blockage (faulty detoxication), delayed?" is that this is unlikely, but the matter is not definitely settled; however, there can never be more alcohol in the blood than the person has ingested. To the third question, "Is the liver or the organism sensitized by the aforementioned drugs, and the alcohol values in the body thereby incorrect?" it is answered that, if the calculation of the blood alcohol level is correct, only 2 possibilities can be seen; either there is a mix-up in the samples, or the statement of the patient is wrong. A confusion of samples can be rectified by checking the numbers on the bottles, and a mistake in establishing the blood alcohol level by repeating the test.

927. Muller, B. P., Tarpey, R. D., Giorgi, A. P., Mirone, L., and Rouke, F. L.
EFFECTS OF ALCOHOL AND MEPHENOXALONE ON PSYCHOPHYSIOLOGICAL TEST PERFORMANCE.
Dis. Nerv. Syst. (Galveston), 25: 373-375 (3 ref.), 1964.
E – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – psychol. perform. – tranquilizers – *CAAAL-11042-J1 A-0948.

40 human subjects received 400 mg mephenoqualone or placebo 4 times/day for 7 days. After 7 days, the subjects were given 3 oz 100 proof vodka, after completing a pre-test battery of psychophysiological tests. 45 min after alcohol, blood alcohol levels were determined, and the psychophysiological tests were again administered. It was found that the difference between mean blood alcohol levels in the drug-alcohol and placebo-alcohol conditions was not statistically significant. The meprobamate-alcohol combination failed to produce a significantly greater decrement in psychophysiological test performance than did alcohol alone. The authors question both the statistical method and representativeness of the test subjects of Zirkle, George A., et al. (J. A. M. A. (Chicago), 173: 1823-1825, 1960).

928. Müller-Limmroth, W.

DIE PHYSIOLOGISCHEN GRUNDLAGEN DER ANFORDERUNG IM STRASSENVERKEHR. [The physiological basis for fitness in road traffic].

In: Wagner, K., et al., eds. *Handbuch der Verkehrsmedizin: unter Berücksichtigung aller Verkehrswissenschaften*. [Handbook of traffic medicine: with consideration of all traffic sciences]. Berlin: Springer, pp. 122-173 (91 ref.), 1968.

G – SEC – review – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mot. vehic. – humans – mammals – CNS – nerv. syst. – stimulants – *CAAAL-0 B-0394.

In a general review of the subject, the author refers to some works on the effect of drugs on the nervous system. A number of published studies have shown that caffeine is not only incapable of neutralizing the alcohol effect, but actually enhances it; in experiments using instrumental conditioning, the reaction of alcohol-treated rats deteriorated further after high doses of caffeine. It can be shown that the CNS amine stimulants have the same effect as caffeine.

929. Müller-Plettenberg, D.

ARZNEIMITTEL UND VERKEHR. [Drugs and traffic].

Ärztliche Mitteilungen (Cologne), 36(19): 1069-1071 (0 ref.), 1962.

G – review – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – mot. perform. – psychol. perform. – species or sex diff. – anesthetics – barbiturates – tranquilizers – *CAAAL-0 A-0949.

A symposium of the Drugs and Traffic Section of the German Association for Automotive Medicine, held in Bad Oeynhausen, March 31-April 1, 1962, is reported. Mention is made of significant interactions in experiments between the variables of alcohol, meprobamate, stability, and sex, with respect to psychomotor performance. Also mentioned is the danger of potentiation between alcohol and barbiturates, and an example is cited in which, 24 hr after inactin narcosis, the ingestion of 1/2 l of beer induced an appreciable potentiation. A variety of other drugs, and their significance in automotive medicine, are discussed.

930. Mundeleer, P.

L'ALCOOL EN ANESTHÉSIE. [Alcohol in anesthesia].

Anesth. Analg. (Paris), 8: 687-690 (0 ref.), 1951.

F – exp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – cardiovasc. – CNS – respir. – analg., antipyret. – autonomic agents – elect., water-bal. agents – *CAAAL-6209-V35 A-0950.

The use of alcohol in conjunction with other anesthetics is described. 27 patients received 10 g luminal 2 hr before, and 10 mg morphine with 0.5 mg atropine 1 hr before surgery. During surgery, the patients received 500 cc isotonic glucose with 10 cc novocaine in 5% sol, and 10-20 cc absolute alcohol in 2-4% sol, at an initial, gradually increasing rate of 40-60 drops/min through a Baxter filter. The advantages of this method include: facilitation of respiration, reduction in the required dose of anesthetic, facilitation of induction of narcosis due to the patient's euphoric state, and acceleration of recovery of consciousness.

931. Munkelt, P., Lienert, G. A., Frahm, M., and Soehring, K.

GESCHLECHTSSPEZIFISCHE WIRKUNGSUNTERSCHIEDE DER KOMBINATION VON ALKOHOL UND MEPROBAMAT AUF PSYCHISCH STABILE UND LABILE VERSUCHSPERSONEN. [Sex-specific differences in effect of alcohol combined with meprobamate on psychologically stable and labile subjects].

Arzneimittelforschung (Aulendorf), 12(11): 1059-1065 (11 ref.), 1962.

G – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – psychot. humans – acute admin. – in vivo – psychol. perform. – species or sex diff. – tranquilizers – *CAAAL-10963-J1 A-0001.

40 male and 40 female students, selected according to their emotional stability or lability, underwent tests involving psychomotor performance, ability to concentrate, and emotional stability-lability, under the following conditions: 800 mg of meprobamate, alcohol (producing 1°/oo in the blood), and meprobamate plus alcohol. Under the influence of meprobamate, the test performance was generally found to be lower in the females. In males, especially in those possessing a high degree of stability, the performance remained unchanged, or was found to increase. Following application of alcohol, the levels of performance and emotional pattern were both found to be reduced, especially in labile individuals. Under the influence of alcohol combined with meprobamate, impairment of sensomotor reactions through alcohol was compensated in the males, whereas the emotional disturbances were increased. In females, the effects were reversed (increased sensomotor impairment, compensation of emotional disturbances). The emotional pattern in stable males was found unchanged after meprobamate-alcohol, as compared with alcohol alone; the emotional pattern of labile males showed further deterioration, following the additional application of meprobamate. In the female group, there was a higher degree of impairment of performance after meprobamate-alcohol in stable than in labile individuals, and a positive change in emotional pattern in stable persons, compared with a negative change in labile persons.

932. Munkelt, P., and Lienert, G. A.
BLUTALKOHOLSPIEGEL UND PSYCHOPHYSISCHE KONSTITUTION. [Blood alcohol level and psychophysical constitution].
 Arzneimittelforschung (Aulendorf), 14: 573-575 (1 ref.), 1964.
 G – ES – FS – SpS – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – psychot. humans – acute admin. – in vivo – blood lev. – psychol. perform. – species or sex diff. – absorp., distrib., stor. – tranquilizers – *CAAAL-0 A-0951.

Controlled experiments were carried out to determine the effect of the blood alcohol level on psychophysical personality factors, in the presence and absence of drugs. In 1 test, the subjects were divided into 2 groups, 1 group receiving 850 mg/kg alcohol in fruit juice plus 400 mg meprobamate, the other receiving alcohol plus placebo. The results in the absence of drugs showed variations in the blood alcohol level of between 0.52 and 1.35°/oo. The psychologically stable subjects had an average blood alcohol level of 0.97°/oo, whereas the labile showed only 0.795°/oo. The sex of the subject had no significant effect. After meprobamate, the absolute blood alcohol level increased in labile subjects, and decreased in the psychologically stable. Conversely, a significant interaction was shown between meprobamate and personality factors in the female group. Meprobamate prolonged absorption time in all subjects.

933. Muraoka, H.
INSHŪ NI YORU JINTAI SHOKINŌ NO HENDŌ TO SORE NI OYOBOSU NI, SAN GAKUHIN NO EIKYŌ. [Functional change of the body at the time of drinking, and the effect of some medicines on it].
 Acta Med. (Igaku Kenkyu) (Fukuoka), 28(3): 287-311 (29 ref.), 1958.
 J – ES – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – acute admin. – in vivo – blood lev. – cardiovasc. – CNS – nutritive agents – tranquilizers – *CAAAL-0 A-1344.

The effects of saki (180 or 540 g po) upon blood pressure, pulse rate, blood alcohol level, hemoglobin content, bound water of the blood, and rectal and axillary temperatures, were compared in alcoholic and non-alcoholic humans (control), and were also determined after injections of d-l methionine, sodium glucuronate, vitamin C, and chlorpromazine to alcoholics. The percentage of bound water was determined by the Kimoto and Tomita vapour pressure method, using sodium chloride (NaCl) or sodium bromide (NaBr) sol in the desiccator. In the non-alcoholics, it was found that, after alcohol administration, the blood pressure fell, the blood alcohol level varied from 0.0775-0.5882 mg/dl, the NaCl bound blood water fell after 1/2 hr and returned to initial value after 1 hr, the NaBr value

showed little change, and the pulse rate and rectal and axillary temperatures increased. Alcoholics showed similar blood pressure and bound water changes; pulse rate increased considerably compared to controls, and in proportion to the amount of alcohol consumed; rectal temperature rose 0.2-0.3°C above that of controls (1°C higher in 1 case); and axillary temperature showed a similar increase. After chlorpromazine administration to alcoholics, it was found that there was a marked decrease in blood pressure; NaCl bound water values decreased, and NaBr values decreased so much (max—1.2%) that there was no return to initial values. The pulse rate increased appreciably. Rectal temperature rose at first, then declined markedly and did not return to initial values; axillary temperatures showed a similar increase.

934. Murphree, H. B., and Price, L. M.
EEG EFFECTS OF BOURBON AND VODKA.
 Fed. Proc. (Bethesda), 24: 517 (0 ref.), 1965.
 E – abst. – congen. stud. – humans – acute admin. – in vivo – blood lev. – mot. perform. – *CAAAL-0 B-0579.

The effects of bourbon, vodka, and distilled water were studied in 10 people, aged 21-56. The problem investigated was whether congeners present in the alcoholic beverages would produce differences in the EEG or other physiological variables. Maximum doses were given (1.0 ml/kg) 3 hr after breakfast. The EEG was recorded for a period of 10 min prior to administration, for the first 30 min after administration, and thereafter for 10 min each half hour up to 3 hr. Electrical recording of eye movement (ENG) was made. Blood alcohol determinations were made by breath analysis. Also, observed effects such as nausea, dizziness, vertigo, etc., were recorded. The results were statistically analyzed. No connection was observed between the ENG, the observed ill-effects, and the blood alcohol levels, and no difference in the EEG effect between bourbon and vodka was revealed by the analysis.

935. Murphree, H. B., and Price, L. M.
COMPUTER TIME-SERIES ANALYSIS OF THE EEG EFFECTS OF ALCOHOLIC BEVERAGES.
 Fed. Proc. (Bethesda), 25: 503 (0 ref.), 1966.
 E – abst. – exp. comp. – congen. stud. – humans – acute admin. – in vivo – mot. perform. – CNS – *CAAAL-0 B-0543.

The EEG effects of 86-proof bourbon and 80-proof vodka were compared with those of a “super bourbon”, 80-proof ethanol with added bourbon congeners. The subjects were 4 men and 5 women aged 22-36. EEG tracings from the left occipital area were recorded for 10 min before administration, for 30 min after administration, and then for 10 min each half hr up to 3 hr. Eye movements were also recorded, and blood alcohol concentration was determined by breath analysis. The results showed that the effects of alcohol alone disappeared quickly, while the effects of the super bourbon lasted much longer, well after blood alcohol concentration had become insignificant.

936. Murphree, H. B., Price, L. M., and Greenberg, L. A.
EFFECT OF CONGENERS IN ALCOHOLIC BEVERAGES ON THE INCIDENCE OF NYSTAGMUS.
 Quart. J. Stud. Alcohol (New Haven), 27(2): 201-213 (26 ref.), 1966.
 E – exp. cont. – exp. comp. – congen. stud. – humans – acute admin. – in vivo – blood lev. – other drug lev. – CNS – nerv. syst. – alcohols – antispasmodics – miscellaneous – *CAAAL-11836-D1 B-0395.

9 healthy humans received, on separate occasions and over a period of 15 min, the following beverages: vodka or bourbon in amounts containing 0.25 and 1 ml/kg ethanol, vodka fortified with congeners

from bourbon (with a congener content equal to 1 quart of bourbon) in an amount containing 0.25 ml/kg ethanol, or water. A nystagmographic recording and blood alcohol determination by breathalyzer were made 15 and 30 min after drinking, and every 1/2 hr up to 3 hr. After the smaller doses of the vodka and bourbon, no nystagmus occurred, but, after the fortified vodka, nystagmus occurred in all subjects for the first hr, and in all but 1 to the third hr. Nystagmus occurred in 5 subjects after the larger doses of both vodka and bourbon. It is noted that, "the total congener content of a quart of the vodka used is about one-third of that in 1 oz of the bourbon whiskey, and the latter caused no nystagmus. In contrast with the extreme nystagmus from the congener content in a quart of bourbon whiskey, therefore, none would be expected to occur from the congeners in a similar amount of the vodka."

937. Murphree, H. B., Greenberg, L. A., and Carroll, R. B.
NEUROPHARMACOLOGICAL EFFECTS OF SUBSTANCES OTHER THAN ETHANOL IN ALCOHOLIC BEVERAGES.
 Fed. Proc. (Bethesda), 26(5): 1468-1473 (14 ref.), 1967.
 E – exp. cont. – exp. comp. – congen. stud. – humans – acute admin. – in vivo – blood lev. – CNS – G.I. tract – nerv. syst. – alcohols – antispasmodics – miscellaneous – *CAAAL-0 B-0396.

A total of 157 experiments were conducted on 28 human subjects who received, on separate occasions, vodka (40%) or bourbon (43%) in amounts corresponding to 1.00 ml/kg absolute ethanol (the bourbon containing 90 times the amount of congeners in the vodka), or a "superbourbon", containing congeners in concentrations of 3.2 to 32 times that contained in the bourbon. The superbourbon was employed to approximate the amount and effects of congener intake during a heavy drinking bout. The effects of the beverages on the blood alcohol concentrations, nystagmus, and electroencephalograms were determined. It was found that the superbourbon produced a more frequent and prolonged incidence of nystagmus than did the bourbon or the vodka. The bourbon produced a greater incidence of drowsiness in the quantitative electroencephalogram than that which occurred with controls. Vodka was intermediate in effect between the bourbon and controls.

938. Murphree, H. B., Greenberg, L. A., and Carroll, R. B.
PHARMACOLOGICAL EFFECTS OF SUBSTANCES OTHER THAN ETHANOL IN ALCOHOLIC BEVERAGES.
 Gastroenterology (Baltimore), 53(5): 759 (0 ref.), 1967.
 E – abst. – congen. stud. – humans – acute admin. – in vivo – psychol. perform. – CNS – *CAAAL-0 B-0963.

A number of common distilled beverages were analyzed by means of gas chromatography to determine their congener content. The results, in order of increasing congener content, were: vodka, gin, blended Canadian whiskey, Scotch whiskey, blended bourbon whiskey, cognac, and straight bourbon. The vodka and bourbon, the latter containing 90 times as much as the former, were selected, together with a "superbourbon" containing still larger amounts of congeners, for a study of congener effects on humans. Duration and intensity of effects were measured according to the production of nystagmus and changes in the electroencephalogram. It was found that congeners do have significant effects which have a longer duration than that of ethanol. These compounds may play a part in the acute and chronic toxicity of some distilled beverages.

939. Murphree, H. B., Greenberg, L. A., and Carroll, R. B.
ELECTROENCEPHALOGRAPHIC AND CARDIOVASCULAR DEPRESSANT EFFECTS OF DIFFERENT ALCOHOLIC BEVERAGES.
 Twenty-eighth International Congress on Alcohol and Alcoholism, Washington, D.C., U.S.A., September 15-20, 1968. Section I, Meeting No. 541, 6 pp. (0 ref.), 1968.
 E – exp. cont. – exp. comp. – presentation – congen. stud. – humans – acute admin. – in vivo – cardiovasc. – metab. proc. – antispasmodics – *CAAAL-0 B-0397.

10 human subjects received, on different occasions, bourbon or vodka in doses equivalent to 1.00 ml/kg absolute ethanol, a superbourbon containing, in the dosages given, 4 or 8 times the amount of congeners found in the bourbon dosages, or orange juice placebo. It was found that the electroencephalographs after high congener-content beverages showed greater and more prolonged incidence of activity. Pulse rates and supine blood pressures were unaffected by bourbon or vodka, but orthostatic systolic and diastolic blood pressures were reduced by both beverages. This action appeared and ended sooner with vodka than with bourbon, but in both cases this occurred well after peak blood alcohol levels. An unexpected finding was that the superbourbon caused a moderate increase in blood pressure in 2 subjects, even with the smaller dose and in the supine position, although neither had any history or evidence of hypertension.

940. Murphree, H. B.
EFFECTS OF ALCOHOLIC BEVERAGES CONTAINING LARGE AND SMALL AMOUNTS OF CONGENERS.

In: Sardesai, V. M., ed. *Biochemical and Clinical Aspects of Alcohol Metabolism*. Springfield, Ill.: Charles C. Thomas, pp. 259-265 (9 ref.), 1969.

E - exp. cont. - exp. comp. - congen. stud. - humans - acute admin. - in vivo - cardiovasc. - CNS
- *CAAAL-0 B-0542.

10 men and women, all moderate social drinkers in good health, were given "monopolar" EEG recordings from left and right frontal, parietal, and occipital areas, with reference to both ears and a ground in the mid-forehead, following which, radial pulse rate and supine and orthostatic blood pressures were recorded. The subjects then consumed bourbon or vodka (in amounts corresponding to 1 ml/kg absolute ethanol), or orange juice placebo, and the recording procedure was repeated hourly until the sixth hr. The findings indicated a prominent fast activity, peaking at 22-23 cycles/sec after bourbon, a result entirely comparable to that found after 1.5 mg/kg thiopental iv, but no detailed statistical evaluation of EEG readings was possible at the time. In cardiovascular tests, placebo had no effect, and pulse rate and supine blood pressure were not significantly affected. Orthostatic blood pressure was affected by both vodka and bourbon; the vodka effect was prominent by the third hr, and disappeared by the sixth hr, whereas the bourbon effect was not prominent until the fifth hr, and probably continued past the sixth hr. The bourbon congeners may have produced a catecholamine release to offset or delay the orthostatic hypotensive effect of ethanol.

941. Murphree, H. B., Schultz, R. E., and Jusko, A. G.
EFFECTS OF HIGH CONGENER INTAKE BY HUMAN SUBJECTS ON THE EEG.

Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 50-61 (11 ref.), 1970.

E - exp. comp. - congen. stud. - humans - acute admin. - in vivo - CNS - *CAAAL-12888-D1
B-0541.

Previous experimental findings concerning congener beverages are reviewed. In the present study, 10 normal volunteers, 8 men and 2 women (21-40 yr), all moderate social drinkers, were used. Baseline EEG recordings were made, following which 1 of the following was given in random order: bourbon or vodka (equalling 1.00 ml ethanol/kg), a superbourbon (equivalent to 0.125 or .0250 ml ethanol/kg), or orange juice placebo. Readings were taken at hourly intervals up to 6 hr. The results showed that, in subjects with large amplitude alpha prior to dose administration, bourbon can produce fast (beta) activity in the frontals of between 16 and 28 cycles/sec, with peak values at 23 cycles/sec; this effect is not apparent after vodka or placebo, and is not seen in the parietals or occipitals. Superbourbon produced a completely different effect from other beverages in 1 subject; the EEG recording 1 hr after dose administration was similar to the baseline readings, but subsequent readings showed a steady diminution of alpha readings, and prominent parietal activity which peaked at 8.5 cycles/sec. Other subjects showed similar alpha variability, associated with drowsiness trends, but the 8.5 cycles/sec parietal activity was a new finding.

942. Mutke, P. H. C.
EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DEN PSYCHOPHYSISCHEN EINFLUSS VON COCA-COLA AUF DEN ALKOHOLBEEINFLUSSTEN MENSCHEN. [Experimental investigations of the psycho-physical effect of Coca-cola on persons intoxicated by alcohol]. Dissertation, Medical Faculty of the University of Heidelberg, West Germany, 32 pp. (44 ref.), 1953.
 G – exp. comp. – DC (unchanged) – humans – acute admin. – in vivo – mot. perform. – psychol. perform. – stimulants – *CAAAL-0 A-0952.

The author carried out a series of comparative experiments to determine the psychophysical effect of Coca-cola (800 cc) in male and female subjects under the influence of alcohol (1400, 2100, and 2800 cc beer with an alcohol content of 3%). The following results were obtained using a precision reaction time apparatus: Coca-cola effected an improvement in the reaction time by +6.2%, and in the Elbelian ring test by +2.1%, with an increase in the error count to -10%. There was no agreement between subjective perceptions and objective test results. According to the author, the obtained improvement in performance is within the limits of chance or variation, and, therefore, may be attributed to chance.

943. Myatt, A. V., and Salmons, J. A.
 CARBON TETRACHLORIDE POISONING.
 A.M.A. Archives of Industrial Hygiene and Occupational Medicine (Chicago), 6(1): 74-82 (38 ref.), 1952.
 E – SEC – DC (add., infra-add., unspec. incr.) – post-mort. – drug-dep. humans – absorp., distrib., stor. – cardiovasc. – G.I. tract – liver, kidney – respir. – anti-infectants – *CAAAL-6364-E3 A-0953.

The symptoms of carbon tetrachloride poisoning and its treatment are described in detail. Of 15 cases reported, 13 patients had a history of alcohol intake prior to exposure to the carbon tetrachloride. The authors state that alcoholics and people in several other categories should not be exposed to carbon tetrachloride. The exact mechanism of increased toxicity of carbon tetrachloride in alcoholics is not known; it may be due to increased absorption of carbon tetrachloride, or to previous damage to liver and kidneys from alcohol.

944. Myers, R. D.
 EFFECTS OF MEPROBAMATE ON ALCOHOL PREFERENCE AND ON THE STRESS OF RESPONSE EXTINCTION IN RATS.
 Psychol. Rep. (Missoula), 8: 385-392 (9 ref.), 1961.
 E – exp. cont. – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – psychol. perform. – tranquilizers – *CAAAL-9384-J2 A-0954.

14 rats received 5% or 20% alcohol sol for 6 months. Lever-pressing performance and preference were tested. It was found that administration of 80 mg/kg meprobamate ip to 4 of the 8 rats receiving 5% alcohol sol caused no change in lever-pressing rates or the preference for alcohol. The administration of meprobamate to 3 of the 6 rats receiving 20% alcohol sol failed to alter lever-pressing performance; these rats, however, always refused alcohol when offered a choice of alcohol, food, or water. When the latter group of rats were exposed to extinction tests in which the reward apparatus was empty, the rats given meprobamate pressed the water lever, whereas those without meprobamate pressed the alcohol lever. Several explanations for the results are proposed and discussed: (a) verification of the failure of meprobamate to cause anorexia, (b) no change in the rate of response and/or general activity level with the meprobamate dosage used, and (c) a decided behavioural effect in shifting the extinction functions from a preponderance of alcohol responses to a majority of water and food responses characteristic of normal animals.

945. Myrsten, A. -L.

EFFECTS OF ALCOHOL AS MODIFIED BY TRANQUILIZERS.

Dissertation, Department of Psychology of the University of Stockholm, Sweden, 34 pp. (40 ref.), 1964.

E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – absorp., distrib., stor. – CNS – tranquilizers – *CAAAL-0 A-0955.

8 normal subjects received, on separate occasions, 0.55 g/kg alcohol po plus 1 of the following: placebo, 800 mg meprobamate po, or 20 mg clonazepam po. Blood alcohol concentration, ocular movements, performance in psychological tasks, subjective intoxication, and other aspects of mood were studied. Meprobamate did not influence the blood alcohol curve; however, in most other variables, it enhanced the alcohol effects. The subjects reported feeling more intoxicated, their performance was more impaired, and their roving ocular movements were increased. After clonazepam, there was a consistent, though slight, difference in the declining phase of the blood alcohol curve, suggesting a change in distribution. The points of maximum disturbance in performance usually occurred later than the maximum blood alcohol values, and, consequently, also later than in the meprobamate or placebo conditions. Performance, on the whole, was less impaired than after alcohol plus meprobamate. The subjects felt less intoxicated than in both other conditions, and the roving ocular movements decreased in intensity, as compared to both other conditions.

946. Myrsten, A. -L.

EFFEKTER AV ALKOHOL PA PSYKOLOGISKA FUNKTIONER. [The effects of alcohol on psychological variables].

Institutet för Maltdrycksforskning [Institute for Research on Malt Beverages], Stockholm, Sweden, Report No. 21, 28 pp. (9 ref.), 1969.

S – exp. cont. – exp. comp. – congen. stud. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – dose resp. – blood lev. – mot. perform. – psychol. perform. – CNS – tranquilizers – *CAAAL-0 B-0544.

The effects on psychological variables of moderate doses of alcohol, alone and in combination with other agents, were studied at the Karolinska Institute in male volunteers. Alcohol and other agents were administered on an empty stomach, and the effects were monitored for 5-10 hr. It was found that, after whiskey intake, the blood alcohol concentration was higher and the self-estimated intoxication was more pronounced, than after beer or wine containing the same amount of absolute alcohol. Administration of 20 mg chlordiazepoxide, 15 min after 0.72 g/kg body wt of alcohol, was observed to improve performance in objective tests, whereas subjective ratings were only slightly changed from placebo conditions. In a similar experiment, 10 mg diazepam produced an increased impairment of performance and more pronounced subjective reactions. The blood alcohol curves were similar after chlordiazepoxide, diazepam, or placebo. Another experiment demonstrated that impairment of performance and self-rated intoxication rose with increased dosage of diazepam. Prophylactic vitamin administration was found to improve performance and decrease subjective intoxication after alcohol administration.

947. McAtee, O. B.

TRANQUILIZING DRUGS, ALCOHOL, AND THE PHYSICIAN'S RESPONSIBILITY.

J. Indiana Med. Ass. (Fort Wayne), 56(8): 1008-1011 (9 ref.), 1963.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – acute admin. – in vivo – blood lev. – CNS – tranquilizers – *CAAAL-0 A-0956.

Experiments were conducted on 24 normal human subjects. In the first test, 200 mg chlorpromazine/-day was administered for 1 week, to establish a working condition. The following experimental conditions were provided: chlorpromazine, alcohol (a sufficient amount to produce a blood alcohol

level of .05%), placebo, or chlorpromazine plus alcohol. It was found that the ascending order of impairment was chlorpromazine, alcohol, and chlorpromazine-alcohol. In the second experiment, an identical group of subjects received 400 mg meprobamate 4 times/day for 1 week prior to the test. Again, 4 test conditions were provided, and the same alcohol dosage used. The ascending order of impairment was meprobamate, alcohol, and meprobamate-alcohol. Various aspects of the problem of tranquilizers, alcohol, and physician responsibility are discussed.

948. McCabe, E. R. B., Layne, E. C., Slusher, N., and Bessman, S. P.
 INTERACTION OF ETHANOL AND GAMMA-BUTYROLACTONE ON SLEEPING
 TIME OF RATS.
 Pharmacologist (Detroit), 12(2): 276 (2 ref.), 1970.
 E – abst. – exp. comp. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – CNS – anesthetics
 – *CAAAL-0 B-0964.

The effects of interaction of gamma-butyrolactone (GBL) and gamma-hydroxybutyrate (GHB) with ethanol on the sleeping time of rats were investigated. Sleeping time was measured by the absence of righting reflex. All agents were given ip, ethanol at a dose of 1.6 ml/100 mg of a 23.75% sol, the GBL at 0.25, 0.33, and 0.41 mmoles/100 g, and a combination of ethanol with the 3 levels of GBL. Sleeping time was significantly prolonged in the "combination" animals, as compared to the animals injected with ethanol or GBL alone. The effect was supra-additive, and was most marked at 0.25 mmoles GBL/100 g. GHB administered with ethanol showed similar interaction.

949. McCall, A. B.
 WHISKEY AND CLAY POULTICE IN SNAKE-BITE.
 Medical Standard (Chicago), 3: 100-101 (0 ref.), 1888.
 E – general – case hist. – DC (antidotal) – humans – cardiovasc. – CNS – *CAAAL-0 A-0957.

The author describes 2 cases of poisonous snake-bite which he personally treated. In the first instance, a 12 yr-old boy was bitten on the ankle by a copperhead snake. Treatment consisted of 100 oz whiskey/24 hr po until improvement was effected, whereupon the dose was diminished. The crisis was over 3-4 days later, and recovery was made in 2 weeks. In the second case, treatment for a finger wound inflicted by a copperhead snake was 1 gallon whiskey/day for 3 days, and a local application of wet clay. Recovery was complete and rapid.

950. McCrea, F. D., and Taylor, H. M.
 THE USE OF PENTAMETHYLENETETRAZOL (METRAZOL) AS A RESPIRATORY
 STIMULANT IN ACUTE ALCOHOLIC DEPRESSION.
 J. Pharmacol. Exp. Ther. (Baltimore), 68: 41-44 (1 ref.), 1940.
 E – exp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – blood lev. – CNS – respir.
 – stimulants – *CAAAL-2397-D2 A-0958.

Ethyl alcohol, in doses from 3 to 7 cc/kg and in 20% aqueous sol, was administered iv to dogs. If respiration did not revive spontaneously after several min of manual respiration, 4.4-7.0 mg/kg metrazol was injected iv. Following the injection, respiration resumed spontaneously within a few sec. 1 animal which received 4.5 cc alcohol required 11.0 mg/kg metrazol to start respiration, and the rate reached a maximum of 210/min. Another dog which received 7.2 cc alcohol/kg was given a total of 18.75 mg/kg metrazol in 3 injections over a period of 31 min. Metrazol had no influence on the rate of disappearance of ethyl alcohol from the blood stream, or on the duration of coma.

951. McCrudden, F. H.
 ÜBER DIE AUSSCHIEDUNG DES MORPHINS UNTER DEM EINFLUSS DEN DARM
 LOKAL REIZENDER STOFFE. [Excretion of morphine under the influence of substances

which locally stimulate the intestines].

Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 62: 374-379 (4 ref.), 1910.

G – exp. – congen. stud. – DC (decrease) – mammals – acute admin. – in vivo – other drug lev. – G.I. tract – analg., antipyret. – *CAAAL-0 A-0959.

In tests on dogs, morphine hydrochloride (0.18 g sc), alone or in combination with 15 cc rum diluted with 15 cc distilled water, was administered repeatedly over a period of time, for a total of 0.990 g morphine (=0.990 g free base). With the morphine-alcohol combination, 61.20% of the injected morphine was recovered in the collected faeces, as opposed to 47.2% in the absence of alcohol. It is conjectured that the etheric oils and other congener components of the rum increased the local stimulating action of alcohol. The author considers that, since it has been shown in past experiments that certain snake venoms are, at least in part, eliminated through the stomach and mucous membrane of the intestine, the possibility of an accelerated elimination of venom under the influence of local irritants is not at all precluded, and that the use of alcoholic beverages in the treatment of snake bites thus may be beneficial.

952. McGee, C. J.

LOWER NEPHRON NEPHROSIS: CARBON TETRACHLORIDE POISONING WITH A REPORT OF THREE CASES.

Amer. J. Med. Sci. (Philadelphia), 218: 636-645 (34 ref.), 1949.

E – SEC – general – DC (add., infra-add., unspec. incr.) – drug-dep. humans – liver, kidney – *CAAAL-0 A-1427.

The characteristics and therapy of lower nephron nephrosis are discussed, with particular reference to 3 cases (2 male, 1 female) of carbon tetrachloride poisoning. In all 3 cases, a chronic use of alcohol occurred prior to the accidental inhalation or ingestion of the poison. 2 of the patients died shortly after admission to hospital, and the third survived and recovered on conservative therapy. Lower nephron nephrosis is classed as a self-limiting disease, in that, if the patient survives, complete recovery follows. The mortality, however, is high—approximately 90%. The advantages and disadvantages of the various treatment methods are pointed out, and the author warns against the altering of water and electrolyte balance of patients with oliguric or anuric kidneys. The frequency of severe lower nephron nephrosis or renal damage following carbon tetrachloride poisoning in the presence of chronic alcoholism is pointed out.

953. McGuire, L. W.

CARBON TETRACHLORIDE POISONING.

J.A.M.A. (Chicago), 99(12): 988-989 (6 ref.), 1932.

E – SEC – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – psychol. perform. – blood comp., sites, lymph – cardiovasc. – CNS – G.I. tract – liver, kidney – respir. – senses – skel., muscle, skin – indust. intox. – *CAAAL-0 A-1345.

The author reports on 7 cases of carbon tetrachloride poisoning that occurred among the employees of a felt-manufacturing plant. The patients were all Italians, and were wine drinkers. The outbreak of poisoning began a few days after the plant started using a 33 1/3% mixture of carbon tetrachloride in the cleaning vats. All of the men complained of nausea and vomiting for from 1-3 days. Other symptoms manifested were headache, diarrhea, vomiting of blood, and burning or smarting about the eyes or mouth. 2 had definite liver enlargements with jaundice, and 4 showed evidence of kidney irritation. One man developed acute nephritis and bronchopneumonia, and was severely ill for 4 months. His drug therapy consisted of epinephrine, caffeine sodiobenzoate, digitalis, and calcium lactate. The author concludes that carbon tetrachloride is a serious poison that most commonly damages the liver and gastrointestinal tract. Calcium deficiency and the use of alcohol render an individual much more susceptible.

954. McKennis, H., Jr., and Haag, H. B.
ON THE CONGENERS OF WHISKEY.
J. Amer. Geriat. Soc. (Baltimore), 7: 848-858 (47 ref.), 1959.
E – exp. – review – congen. stud. – mammals – acute admin. – chronic admin. – in vivo – cardiovasc.
– CNS – liver, kidney – metab. proc. – respir. – indust. intox. – *CAAAL-9462-V2 A-0960.

The literature on congeners is reviewed. The properties of fusel oil and furfural, and the metabolism and effects of ethyl alcohol are discussed. The congeners are necessary for the taste, bouquet, and colour of whiskey, but, when present in large amounts, enhance the possibility of toxic reactions. The evidence suggests that small quantities of congeners in proper balance, such as is found in some types of blended whiskies, may lessen undesirable physiological effects through interaction with ethanol and other constituents of the beverages. A consideration of these factors points to the desirability of further biochemical and toxicological investigations, with emphasis on the fundamental mechanisms.

955. McLean, A. E. M.
COFFEE, ALCOHOL, AND LIVER ENZYMES.
Lancet (London), 2(7576): 1035 (9 ref.), 1968.
E – SEC – exp. comp. – cross-tol. – mammals – acute admin. – in vivo – liver, kidney – metab. proc.
– *CAAAL-0 B-0537.

The author compares barbiturates, coffee, and alcohol as inducers of the liver microsomal hydroxylating enzyme system. The dose of phenobarbitone required to double microsomal hydroxylating activity in rats has been found to be about 3 mg/kg/day, which is equivalent to about 200 mg/day for man. The process achieves half of its maximum after 2 days dosage, and climbs to its maximum (about a 6-fold increase) in a week, after which it remains stable as long as exposure to the inducing drug is continued. The administration of coffee to rats, to give an estimated caffeine dose of 70 mg/kg/day, resulted in no stimulation of drug metabolism after 1 week. When rats were given 20% ethanol in a dose of about 16,000 mg/kg/day, the microsomal enzyme system doubled its activity in 1 week. It is concluded that, in comparison to barbiturates, alcohol (except, perhaps, in alcoholics) and coffee are negligible inducers of microsomal enzyme system activity.

956. McMechan, F. H.
ALCOHOLISM AS A COMPLICATING FACTOR OF ANESTHESIA.
Medical Record (New York), 80: 669-671 (0 ref.), 1911.
E – general – cross-tol. – drug-dep. humans – blood comp., sites, lymph – skel., muscle, skin – analg., antipyret. – anesthetics – autonomic agents – *CAAAL-0 A-0961.

The author states that acute alcoholism contraindicates any operative procedure, except in life-saving emergencies, and that chronic alcoholism demands preliminary medical treatment before any operative interference is advisable. The distinctive value of spinal and regional analgesia for operative procedures is emphasized, and the pros and cons of the gas-oxygen inhalation narcosis, the "ether by the drop" chloroform, and ethyl chloride narcoses are discussed with reference to alcoholic conditions.

957. McQuarrie, D. G., and Fingl, E.
EFFECTS OF SINGLE DOSES AND CHRONIC ADMINISTRATION OF ETHANOL ON EXPERIMENTAL SEIZURES IN MICE.
J. Pharmacol. Exp. Ther. (Baltimore), 124: 264-271 (39 ref.), 1958.
E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – dose resp. – CNS – skel., muscle, skin – anticonvulsants – stimulants – *CAAAL-8793-D2 A-0962.

Quantitative effects of ethanol on thresholds for electroshock and iv pentylenetetrazol convulsions were evaluated for groups of 15-25 mice. Compared with trimethadione and phenobarbital, ethanol was much less potent as an anticonvulsant. Thresholds were increased by doses of ethanol of between 1000 and 4000 mg/kg. To evaluate the effects of chronic administration of ethanol, one group of 50 mice received 1800 mg/kg ethanol, and another group of 15 mice served as control. The mice were examined for neurotoxicity and for protection from electroshock. A high incidence of neurotoxicity reflected the poor protective ability of ethanol. The initial dose of ethanol abolished the hind leg extensor component in 48% of the animals, and caused minimal neurotoxicity in 40% of mice. Single doses of ethanol resulted in transient lowering of seizure threshold, and abrupt cessation of chronic administration of ethanol resulted in hypersusceptibility to seizures for several days.

958. Naalsund, O.
INFLUENCE OF ALCOHOL AS CONTRAINDICATION AGAINST MORPHINE.
 J.A.M.A. (Chicago), 159(7): 727 (0 ref.), 1955.
 E – abst. – general – DC (add., infra-add., unspec. incr.) – drug-dep. humans – blood comp., sites, lymph – CNS – respir. – analg., antipyret. – autonomic agents – *CAAAL-7389-N18 A-0963.

This is an English-language abstract of the article by Odd Naalsund (T. Norsk. Laegeforen., 75: 489-492, 1955). 2 cases of acute morphine intoxication in chronic alcoholics, one fatal, are reported to illustrate the violent symptoms that, because of the synergism between alcohol and morphine-scopolamine, can occur with the use of morphine, in therapeutically allowable doses, in patients affected by alcohol. The influence of alcohol must be considered to be an absolute contraindication for the use of morphine.

959. Naalsund, O.
ALKOHOLPÅVIRKNING SOM KONTRAINDIKASJON MOT MORFIN. [Contraindication of morphine after ingestion of alcohol].
 T. Norsk. Laegeforen. (Oslo), 75: 489-492 (7 ref.), 1955.
 N – general – case hist. – DC (add., infra-add., unspec. incr.) – drug-dep. humans – blood comp., sites, lymph – CNS – respir. – analg., antipyret. – autonomic agents – *CAAAL-7389-N18 A-0964.

2 cases of acute morphine intoxication in chronic alcoholics, one fatal, are reported to illustrate the violent symptoms that, because of the synergism between alcohol and morphine-scopolamine, can occur with the use of morphine, in therapeutically allowable doses, in patients affected by alcohol. The influence of alcohol must be considered to be an absolute contraindication for the use of morphine. The first reported case illustrates the symptomatology in acute morphine intoxication—deep coma, insufficient or suspended respiration with periods of apnea, sometimes of the Cheyne-Stokes type, and marked cyanosis with miosis. Since the introduction of N-allylnormorphine as a specific antidote in acute morphine intoxication, it is especially important to have the symptomatology in mind, so that diagnosis can be made as soon as possible and treatment started. In the first case, a near-fatal condition was entirely changed in about 1 min by the iv injection of N-allylnormorphine hydrobromide.

960. Nadeau, G.
LE MÉTABOLISME DE L'ALCOOL CHEZ L'INDIVIDU NORMAL ET CHEZ L'ALCOOLIQUE CHRONIQUE. [Metabolism of alcohol in the normal person and in the chronic alcoholic].
 Un. Med. Canada (Montreal), 87: 149-152 (21 ref.), 1958.
 F – SEC – review – DC (decrease) – mammals – metab. proc. – elect., water-bal. agents – hormones, hormone antag. – indust. intox. – unclass. ther. agents – *CAAAL-9123-A3 A-0965.

The literature on the subject is reviewed. The rate of metabolism of alcohol is not affected by body temperature, metabolic variations, physical exercise, diet, drugs, or habituation. Doses of dinitrophenol and dinitrocresol, far exceeding those which can be given to humans, have been found to accelerate the rate of alcohol metabolism in animals.

961. Nagy, J.

ALCOHOL-BARBITURATE SYNERGISM.

Acta Morph. Acad. Sci. Hung. (Budapest), Suppl. 8: 61 (0 ref.),

1959.

E – abst. – exp. – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo
– CNS – barbiturates – *CAAAL-0 A-1309.

The author discusses the well-known synergism between alcohol and barbiturates. The effect of dorlotin (amobarbital) is particularly increased by alcohol. In experiments on rats, simultaneous administration of alcohol and dorlotin induced narcosis, whereas either drug given alone in the same dose was ineffective. It was also shown that comparatively harmless doses of the same 2 drugs proved to be lethal when ingested together. The author points out that this latter fact is of clinical significance in determining the cause of such lethal poisonings. In these cases, death is preceded by a prolonged period of unconsciousness, and, during this time, neither alcohol determination nor the estimation of the narcotic drug will give an explanation for the poisoning.

962. Nakanishi, S.

EFFECT OF ORAL HYPOGLYCEMIC AGENTS ON ALCOHOL METABOLISM IN NORMAL AND ALLOXAN-DIABETIC RABBITS. I. EXPERIMENTS WITH NON-DIABETIC RABBITS.

Shinshu Daigaku, Igakubu (Shinshu University Medical Journal) (Matsumoto), 8(3-4): 81-99 (31 ref.),

1963.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin.
– chronic admin. – in vivo – in vitro – dose resp. – blood lev. – metab. proc. – hormones, hormone
antag. – unclass. ther. agents – *CAAAL-0 A-1428.

Carbutamide, tolbutamide, calcium mesoxalate, or sulfadiazine were given to non-diabetic rabbits (100 mg/kg/day po) for 8 weeks. Group A received 2 x 1 IU insulin/kg on the experimental day; both it and the sulfadiazine group served as controls. All animals were fasted for 19 hr prior to iv administration of 1 g/kg 20% ethanol, 1 g/kg glucose in 20% sol, or alcohol plus glucose. In vitro experiments were also conducted on rat liver homogenates. Alcohol significantly lowered the blood sugar level, but did not affect glucose tolerance curves or blood levels of lactate, pyruvate, and alpha ketoglutarate. Glucose and calcium mesoxalate did not affect alcohol metabolism. Carbutamide and tolbutamide did not affect the blood level or metabolic rate of alcohol, but the blood concentration of acetaldehyde in the tolbutamide group was much higher than in controls. In sulfonylurea groups, a significant fall in the serum albumin/globulin ratio, and an increase in gamma globulin were seen; this did not occur after mesoxalate. In rat liver homogenates, low concentrations of sulfonylureas did not affect alcohol dehydrogenase, xanthine, or catalase activity, but higher sulfonylurea concentrations did inhibit alcohol dehydrogenase.

963. Nakanishi, S.

EFFECT OF ORAL HYPOGLYCEMIC AGENTS ON ALCOHOL METABOLISM IN NORMAL AND ALLOXAN-DIABETIC RABBITS. II. EXPERIMENTS WITH ALLOXAN-DIABETIC RABBITS.

Shinshu Daigaku, Igakubu (Shinshu University Medical Journal) (Matsumoto), 8(3.4): 101-118 (10 ref.),

1963.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin.
– chronic admin. – in vivo – dose resp. – blood lev. – metab. proc. – unclass. ther. agents – *CAAAL-0
A-1429.

Alloxan-diabetic rabbits showing glycosuria for more than 4 weeks were selected and divided into severe and moderate diabetic groups on the basis of fasting blood sugar levels. Tolbutamide, carbutamide, or calcium mesoxalate (100 mg/kg/day po) were given for 8 days, following which the responses to iv injections of 1 g/kg 20% ethanol, 1 g/kg glucose in 20% sol, or alcohol plus glucose, were studied. The disappearance of alcohol from the blood in the diabetic rabbits was found to be almost the same as in normal animals. Glucose accelerated alcohol metabolism, but did not affect blood acetaldehyde concentrations. Tolbutamide and carbutamide induced higher blood acetaldehyde concentrations, but did not influence the rate of alcohol metabolism. Calcium mesoxalate had no effect on acetaldehyde or metabolic rates. Blood sugar levels fell after iv alcohol, but glucose tolerance curves were improved. During daily treatment with oral hypoglycemic agents, the fall in blood sugar levels following alcohol injection was less than that before treatment, and the influence of alcohol on iv glucose tolerance curves was less obvious. Effects of alcohol on carbohydrate metabolism of alloxan-diabetic rabbits during long-term drug administration were similar to effects in non-diabetic animals.

964. Nash, H.
PSYCHOLOGICAL EFFECTS AND ALCOHOL-ANTAGONIZING PROPERTIES OF CAFFEINE.
 Quart. J. Stud. Alcohol (New Haven), 27(4): 727-734 (26 ref.), 1966.
 E – exp. – general – DC (decrease) – DC (unchanged) – humans – mot. perform. – psychol. perform.
 – CNS – stimulants – *CAAAL-11838-J1 B-0398.

On the basis of earlier findings, 2 questions are discussed: 1) “Does caffeine enhance or impair human performance?”, and 2) “Do caffeine and coffee antagonize the impairment of human performance by ethyl alcohol?” The experimental evidence is reviewed, and the author concludes that, “The only conclusions that seem warranted are that caffeine-alcohol antagonism possibly varies with psychological function and with dosage levels of the two drugs; that antagonism is possibly absent or negligible under certain conditions; that an exaggeration by caffeine of an alcohol-induced impairment in performance has yet to be demonstrated in human subjects; and that further research is needed clearly to establish the nature and the limits of the caffeine-alcohol antagonism.” Although caffeine may have a limited effectiveness in sobering the intoxicated individual, a serious danger which may arise is that the intoxicated person may be aware of the antagonism by caffeine of certain alcohol-induced impairments of performance, while being unaware of its failure to antagonize the impairment of other, perhaps more critical, aspects of performance.

965. Nasilowski, W.
O WPŁYWIE NIEKTÓRYCH LEKÓW NA PRZEMIANĘ ALKOHOLU ETYLOWEGO.
 [Effects of some drugs upon the metabolism of ethanol].
 Problemy Alkoholizmu (Warsaw), 2/15(11): 1-4 (31 ref.), 1967.
 Po – general – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – blood lev. – CNS – metab. proc. – barbiturates – *CAAAL-0 B-0399.

Literature on the interaction of drugs with ethanol is reviewed and discussed, especially with respect to psychotropic drugs, the use of which is becoming widespread. Types of interaction discussed include interference with ethanol metabolism (e.g., the disulfiram-ethanol type of reaction), the synergistic action of barbiturates, and the interaction of psychotropic drugs with ethanol. Attention is called to the fact that the blood alcohol curve remains stable, even in cases of the simultaneous action of psychotropic drugs and ethanol; this fact is very significant from the medico-legal point of view—the clinical signs of insobriety are often not directly proportional, either to the blood alcohol level or to the blood level of synergistic drugs.

966. Nason, Z. M.
 "SOBERING UP" IS A SERIOUS MATTER.
 Mod. Hosp. (Chicago), 71(5): 98-99 (0 ref.), 1948.
 E – general – DC (antidotal) – DC (add., infra-add., unspec. incr.) – drug-dep. humans – CNS – hormones, hormone antag. – nutritive agents – stimulants – unclass. ther. agents – *CAAAL-5508-Z27 A-0966.

A procedure for "sobering up" the alcoholic, based upon the experiences of treating over 3,000 cases, is given. 2 oz whiskey, diluted with 6 oz of water, is administered every 3 hr for a total of 5 doses; a 6-hr interval is then allowed, and 3 further doses of diluted whiskey are given every 6 hr. In addition, a teaspoon of salt is given after the second and fourth dose. Meanwhile, the patient is given black coffee with sugar. Barbiturates and opiates are not recommended for alcoholics, because they experience a greater narcotizing effect from them.

967. Nathan, P. E., Zare, N. C., Ferneau, E. W., Jr., and Lowenstein, L. M.
 EFFECTS OF CONGENER DIFFERENCES IN ALCOHOLIC BEVERAGES ON THE BEHAVIOUR OF ALCOHOLICS.
 Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 87-100 (7 ref.), 1970.
 E – exp. comp. – congen. stud. – drug-dep. humans – chronic admin. – in vivo – blood lev. – psychol. perform. – CNS – *CAAAL-12729-J1 B-0965.

The behavioural effects of congener differences in various alcoholic beverages were studied in 8 male alcoholics (average age—36 yr) divided into 2 groups which differed only in the order in which beverages were made available. The study lasted 30 days—an 18-day drinking period was preceded by a 6-day withdrawal period. In the first study, the men received 43% ethanol coloured with caramel for the first 9 drinking days, and bourbon for the second 9 days. The second study group drank bourbon for the first 9 drinking days, and vodka for the second 9 days. All subjects peaked at or beyond a blood alcohol level of 200 mg/100 cc during the first days of drinking, then returned to a zero blood alcohol reading 1 or more times during the remainder of the period. It was found that the subjects were more sociable at the start of drinking, and became increasingly depressed and hostile. Neither significant differences in blood alcohol levels, nor beverage preference due to congener differences were noted. The behavioural data were recorded, then tabulated and graphically evaluated. The general conclusion drawn from the results is that differences in the congener content of beverages do not induce consistent differences in behaviour.

968. Neely, E. A.
 PILOCARPIN IN ACUTE ALCOHOLISM.
 Mississippi Valley Medical Monthly (Memphis), 8: 12-18 (1 ref.), 1888.
 E – general – case hist. – DC (antidotal) – humans – cardiovasc. – CNS – G.I. tract – glands – nerv. syst. – skel., muscle, skin – autonomic agents – *CAAAL-0 A-1310.

The use of pilocarpine in the treatment of acute alcoholism is illustrated in 7 case histories. In each case, 1-5 grains of pilocarpine were administered sc, and comparative relief was obtained from the symptoms of alcohol poisoning in about 30 min. The author maintains that the excessive use of alcohol checks the function of secretion, resulting in an accumulation in the blood of nitrogenous waste products which irritate the nervous system and account for the symptoms of acute alcoholism. Pilocarpine, as a powerful motor depressant, relaxes muscular tonus, lowers vascular tension, and stimulates glandular function. In doing so, the drug relieves the patient of the distressing symptoms of high nervous excitement, restless insomnia, and intolerable nausea, and promotes a period of recuperative sleep. It is therefore concluded that pilocarpine is indicated in all cases of acute alcoholism which are characterized primarily by nervous manifestations, tension of the circulatory system, or derangement of the glandular system, and it is contraindicated in cases characterized by depression.

969. Neidhardt, H.
WIRKUNGSÄNDERUNGEN ZWEIER ANALEPTICA DURCH SUBCHRONISCHE ALKOHOLGABEN. [Changes in the effects of two analeptics by subchronical administrations of alcohol].
 Dissertation, Medical Faculty of the University of Hamburg, West Germany, 41 pp. (46 ref.), 1964.
 G – exp. cont. – exp. comp. – DC (decrease) – mammals – chronic admin. – in vivo – dose resp. – antidepressants – *CAAAL-0 A-0967.

Barbiturate narcosis was induced in 60 guinea pigs by administering 20 mg/kg pentobarbital ip. The antagonism of prolintane (Sp 732) and phenylnorcamphane (H 610) was tested in 30 of the animals receiving, for 2 weeks, a 5% alcohol sol, and afterwards a 10% alcohol sol, as the only source of liquid. The control group of 30 animals received tap water instead of the alcohol sol. Both drugs antagonized the barbiturate narcosis in the control group, Sp 732 more than H 610. In the alcohol group, the antagonistic effect was higher than in the controls during the first experimental period (and H 610 was more effective than Sp 732), but it later decreased and finally ceased with the doses of 2 mg and 4 mg/kg. Higher doses of 8 mg to 16 mg/kg maintained the antagonistic effect.

970. Nelemans, F. A.
GENEESMIDDELEN EN VERKEER. [Drugs and traffic].
 Geneeskundige Gids (The Hague), 41(18): 357-359 (0 ref.), 1963.
 D – SEC – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – barbiturates – *CAAAL-0 A-0968.

This article comments on the physician's responsibility to warn his patients of the potential dangers of ingesting certain drugs alone or with ethanol, and then driving a motor vehicle. The drugs which are hazardous in conjunction with ethanol include the barbiturates, amphetamines, antihistamines, and tranquilizers. The barbiturate-alcohol combination is particularly dangerous; after consumption of enough whiskey to produce a blood alcohol level of 0.75°/oo, and enough amobarbital to produce a blood level of 2 mg%, coma will set in. Far too often, the possibility that a patient taking drugs may also use alcohol fails to occur to the physician until it is too late and the patient becomes an accident victim.

971. Nelemans, F. A.
PSYCHOPHARMACA EN ALCOHOL. [Psychopharmacological agents and alcohol].
 Nederl. T. Geneesk. (Amsterdam), 112(27): 1252-1254 (0 ref.), 1968.
 D – exp. – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – blood lev. – tranquilizers – *CAAAL-0 B-0400.

A general discussion is given on the problem of interactions between alcohol and a wide variety of drugs, especially with respect to automotive medicine. The author refers to experiments in which alcohol plus meprobamate induced greater effects in the test subjects than alcohol alone. Tests on animals have revealed a possible synergism between alcohol and iminodibenzyl derivatives and dibenzocycloheptene derivatives; however, the author points out that research is still inadequate in this area. Also mentioned is the fact that stimulants reduce some of the alcohol effects only when the blood alcohol level is less than 1°/oo, and are thus often ineffective as sobering agents.

972. Nelemans, F. A.
PSYCHOPHARMACA IN VERKEER. [Psychopharmacological agents in traffic].
 Nederl. T. Geneesk. (Amsterdam), 112: 1862-1867 (0 ref.), 1968.
 D – SEC – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – dose resp. – *CAAAL-0 B-0401.

The specific properties of various psychopharmacological drugs, their use and abuse, and their effect on psychomotor functions with respect to automotive medicine are reviewed. Also mentioned is the finding in traffic accidents of combinations of psychopharmacological drugs and alcohol (0.35 to 0.40°/oo blood alcohol), and the interaction between lithium compounds and alcohol, phenobarbital, and caffeine. A wide variety of drugs, including neuroleptics, tranquilizers, sedatives, antidepressants, stimulants, and antihistamines are discussed, and the dose-effect relationship underlined. It is suggested that warning labels be issued on all prescriptions, and special instructions given to ambulatory patients who drive.

973. Nelson, G. H., Kinard, F. W., Aull, J. C., Jr., and Hay, M. G.
EFFECT OF AMINOTRIAZOLE ON ALCOHOL METABOLISM AND HEPATIC
ENZYME ACTIVITIES IN SEVERAL SPECIES.
Quart. J. Stud. Alcohol (New Haven), 18: 343-348 (4 ref.), 1957.
E – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo
– in vitro – blood lev. – species or sex diff. – liver, kidney – metab. proc. – *CAAAL-0 A-0969.

Dogs, rats, and mice received 10 g/kg aminotriazole (AT) ip as a 14% aqueous sol. Dogs received 2 g/kg 20% (w/v) alcohol sol iv, and rats and mice received 3 g/kg ip. It was found that AT was effective in reducing hepatic catalase activity, both in vivo and in vitro, in all animals. AT appeared to increase the activity of alcohol dehydrogenase in vivo. The presence of ethanol in the intact animal or in the liver homogenate inhibited the action of AT on catalase activity. Despite the marked reduction in liver catalase activity in dogs receiving injections of AT, the rate of ethanol metabolism was not altered to a significant degree.

974. Nelson, G. H.
ETHANOL PROTECTION AGAINST THE CATALASE-DEPRESSING EFFECT OF
3-AMINO-1, 2, 4-TRIAZOLE.
Science (Washington), 127: 520-521 (10 ref.), 1958.
E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – liver, kidney –
*CAAAL-8283-A2 A-0970.

Ethanol was injected ip in a dose of 1 g/kg, 1 hr before a 1 g/kg ip injection of 3-amino-1, 2, 4-triazole (AT) into rats. The rats were sacrificed at 1/2, 1, and 2 hr after the AT, and the catalase activity in m equivalents/mg liver was measured. Catalase activity was found to be the function of the presence of alcohol in the blood. AT began to take effect before all the alcohol had left the body, and the maximum depressing effects were observed after all ethanol had disappeared. The mechanism of action of ethanol with respect to catalase effects is discussed.

975. Neumann, W., and Walther, R.
BEEINFLUSSUNG DER AUSWERTUNG VON DIGITALISSTOFFEN DURCH
ALKOHOL (VERSUCHE MIT GITOXIGENIN UND GITOXIN). [Influence of alcohol on
the evaluation of digitalis preparations (experiments with gitoxigenin and gitoxin)].
Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 199:
412-420 (10 ref.), 1942.
G – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. –
cardiovasc. – cardiovasc. agents – *CAAAL-0 A-1430.

To investigate the possibility that alcohol, used as a solvent for substances which dissolve with difficulty, may have added to or altered experimental toxicity findings, the effect of alcohol on the toxicities of gitoxigenin and gitoxin, 2 digitalis preparations, were studied in cats. 7 cats (average wt of 2.15 kg) received an infusion of a 1:20,000 saline gitoxigenin sol with 30% alcohol. The LD₅₀ was found to be 0.738 mg/kg; this was compared with previously obtained findings of other researchers

and of the author, of 3.194 mg/kg for a 1:5,000 gitoxigenin sol plus 20% alcohol, and of 3.06 mg/kg for a 1:5,000 gitoxigenin sol plus 5-10% alcohol. The alcohol failed, however, to change the typical electrocardiograph characteristics of gitoxigenin. In a second experiment, the LD₅₀ for a 1:20,000 gitoxin sol, when infused with 30% alcohol and 0.5% pyridine, was found to be 0.75 mg/kg. It is concluded that alcohol, when infused in higher concentrations in a short period of time, can enhance the toxicity of gitoxigenin and gitoxin, resulting in earlier deaths and a lower LD₅₀.

976. Neumeyer, L.

DIE GEGENWIRKUNG VON ALKOHOL GEGEN VERONAL AM FROSCHMUSKEL. [The antagonistic effect of alcohol on veronal in the frog muscle].

Dissertation, Medical Faculty of the University of Göttingen, Germany, 12 pp. (13 ref.), 1936.
G – exp. – DC (decrease) – other org. – in vitro – skel., muscle, skin – barbiturates – *CAAAL-0 A-0971.

On the basis of his finding that small doses of alcohol shift the partition coefficient of veronal between oil and water in favour of the water, the author hypothesized that alcohol will antagonize veronal-induced paralysis. Experimental findings showed that alcohol prevented the veronal from pervading the isolated frog muscle, thus postponing veronal poisoning; after a Ringer bath, recovery was also postponed. The poisoning of the muscle by veronal in Ringer or alcohol-Ringer again showed a postponement of the alcohol effect. An alcohol-Ringer bath of all muscles, however, resulted in an accelerated detoxification of those muscles which were alcohol-treated. In a veronal-poisoned muscle (in pure Ringer), a later alcohol bath postponed recovery. A later alcohol bath accelerated recovery only when the muscle had been treated with alcohol beforehand.

977. New, P. S., Lubash, G. D., Scherr, L., and Rubin, A. L.

ACUTE RENAL FAILURE ASSOCIATED WITH CARBON TETRACHLORIDE INTOXICATION.

J.A.M.A. (Chicago), 181(10): 903-906 (13 ref.), 1962.
E – general – DC (add., infra-add., unspec. incr.) – humans – absorp., distrib., stor. – blood comp., sites, lymph – G.I. tract – liver, kidney – respir. – anti-infectants – *CAAAL-10035-E3 A-0972.

The data from 19 cases, 1 fatal, of acute renal failure associated with carbon tetrachloride (CCl₄) poisoning is reviewed. 17 of the patients were drinking alcoholic beverages at the time of exposure; in 2 cases, the prompt use of CCl₄ as a cleaning agent on clothes worn to a cocktail party the same evening was sufficient to cause acute renal failure. Further comments on the relationship of concomitant alcohol intake with CCl₄ intoxication are made with respect to present experimental evidence.

978. Newell, G. W., Shellenberger, T. E., and Reinke, D. R.

CHRONIC EFFECTS OF ALCOHOL, MUSCATEL, AND SHERRY ON THE GROWTH AND PERFORMANCE OF MALE RATS.

Toxic. Appl. Pharmacol. (New York), 6: 696-700 (11 ref.), 1964.
E – exp. cont. – exp. comp. – congen. stud. – mammals – chronic admin. – in vivo – mot. perform. – CNS – liver, kidney – nerv. syst. – *CAAAL-11073-D2 A-1431.

To determine whether the prolonged ingestion of ethanol or inexpensive and expensive kinds of sherry or muscatel (each sherry or muscatel sol being composed of 8 commercial wine brands) adversely affected liver function, neuromuscular coordination, or CNS sensitivity, newly-weaned male rats were given 12% alcohol or wine sol over a 32-week period. Wt gain for the period averaged about 10% less than in control animals given water; this may have been due to a much lower fluid consumption by the alcohol and wine groups. No gross abnormalities were noted at necropsy. Wt of liver, kidney, spleen, heart, and testes of test groups were not significantly different from water controls. A slight fatty infiltration of the liver was observed in alcohol and wine groups, otherwise no micropathology

was found. Bromosulfalein, audiogenic seizure, and electroshock were the same for control and experimental animals. In the rotating bar test for neuromuscular coordination, control rats remained longer on the bar than did alcohol or wine groups, the difference apparently being due to alcohol, rather than to any other wine constituent.

979. Newman, H. W., and Cutting, W. C.
 THE ACTION OF DINITROPHENOL AND INSULIN IN ACCELERATING THE
 METABOLISM OF ETHYL ALCOHOL.
 J. Clin. Invest. (Boston), 14: 945-948 (15 ref.), 1935.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – humans – mammals – acute admin.
 – chronic admin. – in vivo – in vitro – liver, kidney – metab. proc. – respir. – hormones, hormone
 antag. – *CAAAL-1128-A1 A-0973.

The effects of both acute (500 mg) and chronic (100 mg, 3 times/day for 2 weeks) administration of dinitrophenol upon the rate of disappearance of ethanol from the blood was investigated in humans. It was found not to be an effective accelerator at safe dosages. Insulin and insulin-free pancreatic extract were capable of increasing the rate of alcohol metabolism by approximately 50% in therapeutic doses in man. Dinitrophenol accelerated the metabolism of alcohol in rat liver slices, and insulin and insulin-free pancreatic extract affected the oxidation of alcohol in vitro in the absence of animal tissue.

980. Newman, H. W., Cutting, W. C., and Tainter, M. L.
 ACTION OF DINITROPHENOL ON RATE OF OXIDATION OF ETHYL ALCOHOL IN
 VITRO.
 Proc. Soc. Exp. Biol. Med. (New York), 32: 1479-1480 (6 ref.), 1935.
 E – exp. cont. – DC (decrease) – mammals – in vitro – liver, kidney – metab. proc. –
 *CAAAL-1130-A2 A-0974.

The action of dinitrophenol on the rate of oxidation of ethanol was investigated in rat liver slices. At concentrations from 1:5,000,000 to 1:20,000,000, dinitrophenol slightly increased the rate of oxidation, while higher concentrations slightly diminished it. This indicated that, under some conditions, an increase in tissue metabolism produced by dinitrophenol is accompanied by an increased rate of oxidation of alcohol. From this it may be deduced that the increased rate of fall of blood alcohol concentration caused by dinitrophenol in animals may be accounted for, in part at least, by an increased rate of its oxidation in the tissues.

981. Newman, H. W., and Tainter, M. L.
 EFFECT OF DINITROPHENOL ON RATE OF ALCOHOL METABOLISM.
 J. Pharmacol. Exp. Ther. (Baltimore), 57: 67-73 (9 ref.), 1936.
 E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – respir. –
 *CAAAL-1113-A6 A-0975.

The effect of dinitrophenol (6-10 mg/kg) upon the rate of disappearance of alcohol from the blood was investigated in dogs. When elimination of alcohol from the lungs was prevented by rebreathing, the hyperventilation of dinitrophenol did not modify the rate of decline of blood alcohol, but, when the dogs breathed into room air, alcohol elimination was doubled. A similar degree of hyperventilation, produced mechanically, also resulted in an increased rate of decline of blood alcohol. Therefore, the authors consider the increased rate of disappearance due to the hyperventilation, and not to an increased rate of metabolism or the increased temperature.

982. Newman, H. W., and Richardson, A. P.
 THE EFFECT OF ALCOHOL ON PENETRATION OF BISMUTH INTO THE CENTRAL NERVOUS SYSTEM.
 American Journal of Syphilis, Gonorrhea and Venereal Diseases (St. Louis), 21: 77-80 (10 ref.), 1937.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – CNS – *CAAAL-0 A-0976.

The therapeutic equivalent dosage for man, 0.05 cc/kg of iodobismutol, was administered im to 6 dogs twice weekly for 3 weeks. 3 of the dogs also received 10 cc/kg of ethanol daily. All of the dogs receiving alcohol plus iodobismutol showed a higher bismuth concentration in the brain. The mortality rate for alcoholized dogs was 75%, as against 25% for the non-alcoholized dogs. Whether this increased mortality was due to increased toxicity of the bismuth because of increased penetration, or merely to the cumulative toxic action of the 2 drugs, is impossible to determine in the light of present knowledge. The possible significance of these results for the treatment of syphilis is discussed.

983. Newman, H. W.
ACUTE ALCOHOLIC INTOXICATION: A CRITICAL REVIEW.
 Stanford: University Press, 207 pp. (14-15, 61-65, 95-96, 123-124, 142-143, 182-195) (199 ref.), 1941.
 E – review – cross-tol. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – drug-dep. humans – mammals – acute admin. – chronic admin. – in vivo – in vitro – dose resp. – blood lev. – other drug lev. – absorp., distrib., stor. – CNS – G.I. tract – liver, kidney – metab. proc. – respir. – amphetamines – anesthetics – autonomic agents – barbiturates – stimulants – *CAAAL-82-A3 A-0977.

A critical review of the literature, chiefly from the biological and medical viewpoint, is presented. The relevant ethanol interaction sections include: the influence of sympathomimetic substances on alcohol absorption, the role of various drugs and oxygen in alcohol metabolism, the effects of strychnine, barbiturates, and oxygen on lethal alcohol concentrations, and the methods of treatment of alcohol-induced central nervous system depression.

984. Newman, H. W., and Newman, E. J.
 FAILURE OF DEXEDRINE AND CAFFEINE AS PRACTICAL ANTAGONISTS OF THE DEPRESSANT EFFECT OF ETHYL ALCOHOL IN MAN.
 Quart. J. Stud. Alcohol (New Haven), 17(3): 406-410 (4 ref.), 1956.
 E – exp. cont. – exp. comp. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – mot. perform. – CNS – senses – amphetamines – stimulants – *CAAAL-7521-D1 A-0978.

6 subjects were given 0.1 cc of alcohol/lb at 20 min intervals. Dexedrine (15 mg) and caffeine (300 mg) were administered 45 min before the alcohol. A battery of tests revealed that dexedrine and caffeine were ineffective in combatting the depressant effect of alcohol on the nervous system, when used in ordinary therapeutic dosage. In none of the subjects was there evidence of significant elevation of the concentration of alcohol in the blood at which failure on the tests occurred. The most consistent results, with respect to the blood alcohol concentration at which failure occurred, were obtained with the balance test; all 4 subjects who received alcohol and caffeine failed at a higher blood alcohol concentration than with alcohol alone, but in no case was the increase more than 13 mg/cc, or about 10% of the total value. Even if the small differences recorded are statistically significant, they are not of practical value, since an improvement of 50% would be necessary to increase the tolerance of the less tolerant subjects to that of the 2 most tolerant.

985. Nichols, J. L.

THE EFFECTS OF DRUGS ON DRIVING RELATED BEHAVIOR AND THE IMPACT OF DRUGS ON HIGHWAY INCIDENTS.

National Highway Safety Bureau, Office of Alcohol Countermeasures, Traffic Safety Programs, Washington, D.C., 90 pp. (91 ref.), September 4, 1970.

E – SEC – general – review – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – drug-dep. humans – CNS – barbiturates – hallucinogens – sed., hypnot. – stimulants – tranquilizers – *CAAAL-0 B-0966.

In an extensive review and analysis of the present situation regarding the effects of drug use on driving behaviour and traffic accidents, the author considers the extent of drug use in the American culture, the effects of drugs on the human body, experimental and laboratory investigations, the assessment of risk, and the incidence of drug use in highway situations. The report ends with several conclusions based on the evidence, and 6 recommendations are presented. U.S. federal narcotic and dangerous drug laws, a drug use glossary, and a summary of research investigations, are presented in appendices. The interaction of alcohol with other drugs is considered, with respect to depressants, stimulants, hallucinogens, and narcotics.

986. Niedeggen, G.

CORAMIN INTRAMUSKULÄR BEI ALKOHOLVERGIFTUNGEN. [Intramuscular coramine in alcohol intoxication].

Munchen. Med. Wschr. (Munich), 86(23): 893-894 (1 ref.),

1939.

G – general – DC (antidotal) – humans – CNS – G.I. tract – stimulants – *CAAAL-947-N13

A-0979.

The author reports on his clinical experience with about 30 cases of acute alcohol intoxication, in which im injections of coramine (1.7 to 5.5 cc) were used to sober up the patients. After 10 to 15 min, the patient could think more clearly, his answers were correct, and his agitation disappeared. Vomiting often ceased promptly. The author emphasizes that the method of coramine injection has also the great advantage that, in cases of emergency, a nurse can administer the injection before the arrival of a physician.

987. Nielsen, G. L.

CALIFORNIA'S SINGLE VEHICLE FATALITY STUDY: ALCOHOL, DRUG AND ORGANIC FACTORS.

National Coroners Association Conference, Miami Beach, Florida, July 26-29, 8 pp. (0 ref.),

1966.

E – presentation – stat. surv. – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev. – tranquilizers – *CAAAL-12324-D1 B-0402.

In this unpublished report, extensive data on fatal single vehicle accidents are presented. Of 871 cases in which death of the driver occurred instantaneously or within 15 min, 647 (74.3%) had been drinking, 113 (12.9%) were under the influence of drugs, and 78 (8.96%) were under the influence of both alcohol and drugs. The author points out the importance of the problem of alcohol-drug interaction in driving, and the need for more detailed study of this question. In addition, 155 cases in which death precipitated the accident are discussed.

988. Nielsen Kudsk, F.

THE INFLUENCE OF ETHYL ALCOHOL ON THE ABSORPTION OF MERCURY VAPOUR FROM THE LUNGS IN MAN.

Acta Pharmacol. (Copenhagen), 23: 263-274 (14 ref.),

1965.

E – exp. cont. – DC (decrease) – humans – acute admin. – blood lev. – absorp., distrib., stor. – metab. proc. – respir. – miscellaneous – *CAAAL-12324-D1 B-0403.

Mercury vapour absorption in 2 men dropped from about 84% to about 65%, following ingestion of 20 to 24 g of absolute alcohol diluted in soda water, and from 75% to 87% to between 49% and 63% in 3 men who drank 57 g of alcohol. The greatest blood alcohol concentration coincided with maximum inhibition of mercury absorption in all cases. The intake of food caused a secondary inhibition. Further experiments confirmed that ingestion of moderate amounts of alcohol, with or between normal meals, causes prolonged and appreciable reduction in absorption of mercury vapour from the lungs; normal eating, without alcohol, had no effect on mercury absorption.

989. Nieschulz, O.

DIE ENTHEMMENDE WIRKUNG DES ALKOHOLS UND DEREN BEEINFLUSSUNG DURCH PSYCHOPHARMAKA IN VERSUCHEN MIT MÄUSEN UND RATTEN.

[Disinhibiting effect of alcohol and the influence of psychopharmacological drugs thereon in experiments with mice and rats].

Rostock Universität. Wissenschaftliche Zeitschrift. Mathematisch-Naturwissenschaftliche Reihe (Rostock), 15(5): 725-729 (5 ref.), 1966.

G – ES – FS – RS – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – psychol. perform. – barbiturates – stimulants – tranquilizers – *CAAAL-0 B-0404.

Groups of 6-15 rats were tested for avoidance behaviour. Alcohol (5 ml 25% alcohol/kg) and chlorpromazine (5 mg/kg) each decreased vigilance by 9% and 15%, respectively, and, in combination, by 43%. Perazine (10 mg/kg) had little effect alone, but, in combination with ethanol, resulted in a 30% reduction; phenobarbital had a similar effect, whereas caffeine partly counteracted the alcohol effect. Part of the floor leading to the feeding box was covered with sandpaper, and the degree of hesitation was assessed. The results were: 37% hesitation 30 min after alcohol (above dosage), 2% 60 min after 7.5 ml 25% alcohol/kg, and 70% 60 min after perazine (10 mg/kg) and alcohol (7.5 ml/kg) in combination. Learning time was evaluated in groups of 30-65 mice. There was an average of 5 repetitions without drugs, 7 repetitions 60 min after 10 ml 25% alcohol/kg, and 10 repetitions after 15 ml 25% alcohol/kg; both the alcohol and perazine (25 mg/kg) made the mice livelier and more excited, but a combination of the two failed to intensify the effect of each given singly. Chlorpromazine (2.5 mg/kg) failed to affect learning capacity, but significantly affected it in combination with alcohol.

990. Nieschulz, O.

DIE BEEINFLUSSUNG DES VERHALTENS VON RATTEN IN EINER KONFLIKTSITUATION DURCH

10-[3'-(4''-METHYL-PIPERAZINYL-1''-PROPYL-1')]-PHENOTHIAZIN UND ALKOHOL.

[Influence on the behavior of rats by 10-[3'-(4''-methyl-piperazanyl-1''-propyl-1')]-phenothiazine and alcohol in a conflict situation.].

Arzneimittelforschung (Aulendorf), 17(2): 190-193 (26 ref.),

1967.

G – ES – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – psychol. perform. – tranquilizers – *CAAAL-0 B-0405.

The combined influence of phenothiazines and alcohol on fear and anxiety was investigated. For hungry rats which had to work a lever to obtain food, a conflict situation was devised (easier access to food was combined with electric shock). In the conflict situation, the behaviour changed as compared with the normal situation. By omitting the stimuli, the trained-conflict behaviour became less consistent. The time taken to press the lever was significantly longer after perazine (10 mg/kg) and after alcohol (1.25 ml/kg) than in the control group. Simultaneous administration of both substances did not enhance the effect. The effects can be considered as an inhibition of fear and anxiety.

991. Niggemeier, K.
ÜBER DIE BEEINFLUSSUNG DER VERGIFTUNGEN MIT NITROTOLUOL, DINITROTOLUOL, NITROPHENOL, DINITROPHENOL, ORTHONITRANISOL UND ANILIN DURCH ALKOHOL. [The influence of alcohol on poisonings by nitrotoluol, dinitrotoluol, nitrophenol, dinitrophenol, orthonitranisol, and aniline].
 Dissertation, Medical Faculty of the University of Würzburg, Germany, 36 pp. (0 ref.), 1903.
 G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – miscellaneous – *CAAAL-0 A-0980.

In controlled experiments on rabbits, all animals survived nitrotoluene poisoning in doses of up to 2.0 cc/kg by stomach tube in the presence or absence of alcohol (3.5 cc/kg by stomach tube). The animals died after 0.5 g/kg dinitrotoluene, but survived lower doses (90.3 g/kg) in the presence or absence of alcohol. Nitrophenol (0.5 g/kg) and dinitrophenol (0.132 g) induced rapid death following administration of alcohol in the above dosage. Alcohol increased the toxic effects of orthonitranisol (0.5 cc) to an appreciable degree. At a dose of 0.7 cc orthonitranisol/kg, the alcohol-treated animal died, whereas the untreated animal recovered; at a dose of 1.0 cc/kg, the alcohol-treated animal died significantly sooner than the untreated animal. Moreover, the alcohol-treated animals were much more affected by aniline than the untreated animals. In the presence of alcohol, the animal given an aniline dose of 1.0 cc/kg died 10 hr later.

992. Nikki, P., and Vapaatalo, H.
INFLUENCE OF ETHANOL ON HALOTHANE-ANAESTHETIZED RATS.
 Scand. J. Clin. Lab. Invest. (Oslo), 25 (Suppl. 113): 94 (0 ref.), 1970.
 E – abst. – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – anesthetics – *CAAAL-0 B-0967.

The effects of ethanol on body temperature, shivering, and brain, heart, and adrenal gland tissue monoamines were investigated in halothane-anesthetized rats. Ethanol, either 1.5 or 2.5 g/kg ip, was administered in combination with 30 min of 2% halothane. Rectal temperature was measured with a thermocouple, shivering with an electronic device, and tissue monoamines spectrophotofluorometrically. Ethanol slightly increased body temperature in unanesthetized rats, the 2.5 g/kg dose more than the 1.5 g/kg dose. The larger ethanol dose produced a greater decrease of temperature in halothane-anesthetized rats than did the smaller one. The return of normothermia was delayed after both ethanol doses, but shivering was prevented after the larger one. Halothane elevated brain dopamine concentrations in the control group and in the group pretreated with 2.5 g/kg ethanol; the latter group showed a decreased concentration of brain 5-hydroxytryptamine. No other changes of monoamines in brain or other tissues were found. It is suggested that ethanol may have central mechanisms affecting the temperature regulation, mediated through brain monoamines.

993. Nishioka, T.
HABU-DOKU NO YAKKŌGAKU-TEKI KENKYŪ. [Pharmacological studies on trimeresurus venom].
 Kagoshima Daigaku Igaku Zasshi (Kagoshima University Medical Journal) (Kagoshima), 12(6): 112-156 (44 ref.), 1961.
 J – SEC – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – mot. perform. – *CAAAL-9713-D2 A-0981.

The effect of trimeresurus venom on various drugs was studied in mice and in *Rhynchelmis orientalis*. Venom toxicity was decreased in *Rhynchelmis orientalis* given chlorpromazine (0.005%), chloral hydrate (1.5%), or ethanol (25%). Ethanol (0.025 to 0.15 ml of 50% sol/g) was among the drugs that decreased toxicity in mice. Death by electroshock was antagonized by the combination of any of the drugs tested with venom. Increased kinetic activity, as seen with swimming tests, was observed with all tested drugs combined with venom.

994. Nobes, P.
 INTRAVENOUS BARBITURATES FOR DRUNKENNESS.
 Brit. Med. J. (London), 1: 836 (2 ref.), 1953.
 E – general – DC (antidotal) – humans – G.I. tract – gastrointest. agents – sed., hypnot. –
 *CAAAL-6478-N4 A-0982.

The author criticizes the treatment for calming excited inebriates advocated by Bartley, A. H. (Brit. Med. J. (London), 1: 163, 1953) as potentially dangerous, since thiopentone given to a patient, without a thorough stomach wash-out, may result in the “silent regurgitation” of gastric contents. A far safer, if less dramatic method of calming the obstreperous drunk is the administration of 10-20 ml paraldehyde im. Subemetic doses of apomorphine have also been found effective.

995. Nostiz, H.
 DIE WIRKUNG VON CHININ AUF DIE ALKOHOLKONZENTRATION DES BLUTES. [The effect of quinine on the alcohol concentration of the blood].
 Dissertation, Medical Academy of Düsseldorf, Germany, 19 pp. (19 ref.), 1938.
 G – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – other drug lev. –
 absorp., distrib., stor. – anti-infectants – *CAAAL-0 A-0983.

In each of 4 trials, the investigator ingested 275 g of rye whiskey; in trials b, c, and d, 1.0 g of quinine was taken in addition (once simultaneously with alcohol, once 1/2 hr before, and once 1/2 hr after the alcohol). Results showed that quinine in the doses used had no noticeable effect on the blood alcohol concentration. The accelerated alcohol absorption in trials b, c, and d was ascribed to causes other than the influence of quinine, conceivably to a general fluid increase of the body, or to a general increase of absorption due to prolonged alcohol intake.

996. Notz-Schwarz, I. von
 ÜBER DEN VERLAUF DER BLUTALKOHOLKURVEN BEI VERABREICHUNG VON COFFEIN, CARDIAZOL, PYRAMIDON UND INSULIN. [The course of the blood alcohol curve after administration of caffeine, cardiazol, pyramidon, and insulin].
 Dissertation, Medical Faculty of the University of Giessen, Germany, 22 pp. (17 ref.), 1938.
 G – exp. comp. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – metab. proc. –
 analg., antipyret. – hormones, hormone antag. – stimulants – *CAAAL-0 A-0984.

Tests were carried out on 2 women and 3 men to measure the effect of various substances on the blood alcohol curve, using the Widmark method. Alcohol was administered po in a ratio of 0.5 g/kg in 100 cc distilled water. The following substances were administered immediately before alcohol ingestion: 0.2 g pure caffeine in 10 cc distilled water, liquid cardiazol (20 drops in a 10% sol), pyramidon (3 tablets, 0.1 g dissolved in 10 cc distilled water), and 10 IU insulin sc. Blood samples were taken 20, 40, 60, 90, 120, 140, 160, 180, and 210 min after the beginning of the experiment. The results showed, except for pyramidon, a diminution of subjective and objective intoxication and of hangover phenomena, the diminution being most pronounced with caffeine and insulin. The curve of the blood alcohol values, except for insulin, was not affected by the substances. Insulin showed a tendency to accelerate the rate of metabolism of alcohol, as indicated by lower blood alcohol values.

997. Novi, M.
 IL SINERGISMO DELL'ALCOOL E DELLA LECITINA CON L'ETERE NELLA NARCOSI CHIRURGICA. [Synergism of alcohol and lecithin with ether in surgical narcosis].
 Clinica Chirurgica (Milan), 31: 105-130 (14 ref.), 1928.
 I – exp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – CNS – anesthetics –
 *CAAAL-0 A-1432.

6 series of experiments were performed on male and female patients awaiting surgery, to investigate the use of lecithin, alcohol, or their combination, in facilitating ether narcosis. The 6 series comprised the following administrations prior to ether narcosis: injection of 1 cg morphine chlorate, 1 cg morphine + injection of 3 cc commercial lecithin, 1 cg morphine + 10 cc ethanol and .005 g saccharin in 10 cc water po, 1 cg morphine + ethanol + 4.5 cc lecithin, 1 cg morphine + injection of 30 cg pure lecithin, or 20 cg pure lecithin. The time required for onset of narcosis, ether dosage necessary to maintain narcosis, and speed of inhalation were recorded and tabulated. It was found that alcohol decreased the dose of ether necessary to reach narcosis in 10 min, but not as effectively as did commercial lecithin. Alcohol in combination with commercial lecithin was more effective in lowering the narcotic dose and shortening the inhalation period than was alcohol alone, but the combination was not effective as the lecithin alone. The commercial egg lecithin was found more effective than pure lecithin. It is concluded that lecithin, alone or in combination with ethanol, as an addition to morphine, facilitates surgical ether narcosis, and has important applications.

998. Nukada, T., and Andoh, N.

ETHYL ALCOHOL INHIBITION OF BRAIN MITOCHONDRIAL RESPIRATION
STIMULATED BY DINITROPHENOL.

Jap. J. Pharmacol. (Kyoto), 17(2): 325-326 (2 ref.),

1967.

E - exp. cont. - DC (decrease) - mammals - in vitro - CNS - *CAAAL-0

B-0406.

The inhibitory action of alcohol on brain mitochondrial respiration stimulated by 2,4-dinitrophenol (DNP) was investigated. DNP or alcohol had no effect on the respiration of rat brain mitochondria, when the mitochondria were incubated in a complete medium containing 6.7 mM magnesium sulphate, 22 mM sodium pyruvate, 0.67 mM sodium maleate, and 0.3 mM nicotinamide adenine dinucleotide in a vol of 3 ml. But, when rat brain mitochondria were incubated in a simple medium which contained 50 mM potassium chloride, 20 mM tris-buffer at pH 7.4, 6.7 mM magnesium sulphate, 6.7 mM sodium pyruvate, and 0.67 mM sodium malate, the addition of DNP increased the respiration remarkably. The respiration, stimulated by the addition of DNP in a simple medium, was inhibited significantly by the addition of alcohol (at concentrations which otherwise little affected the unstimulated brain mitochondrial respiration).

999. Nyiogi, S. K., and Lu, F. C.

THE COMBINED ACTIONS OF ALCOHOL AND SECOBARBITAL.

Canadian Society of Forensic Science, Proceedings (Ottawa), 3: 109-117 (18 ref.),

1964.

E - exp. cont. - DC (add., infra-add., unspec. incr.) - mammals - acute admin. - chronic admin. - in vivo - dose resp. - blood lev. - other drug lev. - CNS - liver, kidney - metab. proc. - barbiturates - *CAAAL-0

A-0985.

The effect of repeated treatment with alcohol on secobarbital-induced sleeping time (10 rats were given alcohol, 3 g/kg po once daily for 8 days, while controls received water; on the ninth day, secobarbital was injected into both groups) and on the LD₅₀ of secobarbital (in each of 4 groups of rats, one control without pretreatment, and the others receiving respectively: water, alcohol (3 g/kg), and alcohol (5 g/kg), po for 8 days), 4 doses of secobarbital were given, the percentage mortality at each dose converted to probit, and the LD₅₀ estimated. It is concluded that alcohol augments the action of secobarbital above the 0.05% blood alcohol level, that alcohol does not impede secobarbital metabolism, and that repeated administration of alcohol does not alter the hypnotic or lethal activity of secobarbital.

1000. Oardă, M., and Mihăilescu, M.

ACHIZIȚII NOI ÎN TRATAMENTUL INTOXICAȚIILOR ACUTE. [New findings in the treatment of acute intoxications].

Viața Medicală (Bucharest), 11(2): 123-128 (33 ref.),

1964.

Ru – SEC – general – DC (antidotal) – humans – CNS – hormones, hormone antag. – stimulants
– *CAAAL-0 A-0986.

This article is a summary of substances which are the main cause of the increased incidence of poisonings. Treatments are described. The intoxicants include: barbiturates, morphine and its derivatives, tranquilizers, chlorpromazine, antihistamines, hydrazides, alkyl-phosphates, and salicylic derivatives. Their pharmacodynamic properties are listed as well. For treatment of alcoholic comas, either 30-50 mg of ritalin iv or 200 mg of triiodothyronine iv is recommended.

1001. Oberlandesgericht Celle [Superior District Appeal Court, Celle]
URTEIL VOM 6.6.1963. [Judgement delivered June 6, 1963].
Neue Juristische Wochenschrift (Berlin), 16: 2385-2386 (16 ref.), 1963.
G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev.
– analg., antipyret. – *CAAAL-0 A-0987.

A court decision, regarding impaired driving as a result of combined intake of alcohol and butazolidin, is reported. The accused pleaded in his defense that he was unable to recognize his condition because of the additive effect of the drug (blood alcohol level 1.47 g°/oo). Moreover, his physician gave him no instructions to abstain from alcohol while under drug treatment. It remained open to question whether the amount of alcohol ingested by the defendant would by itself impair the driving ability, and thus render him liable. Leniency was shown by the court, on the assumption that the driving impairment may have been contributed to in part by the intake of butazolidin. A number of precedents are cited involving the influence of alcohol and smoking.

1002. Oberlandesgericht Frankfurt [Superior District Appeal Court, Frankfurt]
URTEIL VOM 28.4.65. [Judgement delivered April 28, 1965].
Deutsches Autorecht (Munich), 35: 106-108 (26 ref.), 1966.
G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev.
– tranquilizers – *CAAAL-0 B-0407.

A case of impaired driving was appealed. The defense pleaded diminished responsibility because, although the blood alcohol concentration was 2.0°/oo, the alcohol effects were potentiated by the intake of 1 capsule of neopyrine (for a cold) and 5 capsules of restenil (for cardiovascular discomfort). The conviction was upheld, since the court concluded that it is the duty of every driver to guard against and notice unusual reactions, and to stop driving when such reactions occur. This the accused failed to do.

1003. Oberlandesgericht Hamburg [Superior District Appeal Court, Hamburg]
BESCHLUSS VOM 11.8.1964. [Decision rendered August 11, 1964].
Deutsches Autorecht (Munich), 34: 27-28 (2 ref.), 1965.
G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev.
– barbiturates – *CAAAL-0 B-0408.

A case of impaired driving involving synergism between alcohol and barbiturates (blood alcohol level 2.4°/oo) is reported. The defendant was found guilty as charged, inasmuch as the alcohol consumed by him was enough to cause impairment, notwithstanding the additive effect of the barbiturates. The particular barbiturates taken are not identified.

1004. Oberlandesgericht Hamm [Superior District Appeal Court, Hamm]
URTEIL VOM 25.2.60. [Judgment delivered February 25, 1960].
Deutsches Autorecht (Munich), 29: 235-236 (0 ref.), 1960.

G – general – DC (add., infra-add., unspec. incr.) – DC (unchanged) – med.-leg. – mot. vehic. – humans – *CAAAL-0 A-0988.

A court decision regarding a case of impaired driving is reported. The defendant, who ingested some alcohol in the course of the day, participated in a wine-sampling party later in the evening, consuming about 4-5 glasses of wine. The court denied leniency to the accused, who pleaded that the sudden loss of consciousness was due to having combined with the alcohol intake the smoking of a cigar (to both of which he was unaccustomed). The court did not question the fact that loss of consciousness occurred; nevertheless, the accused was found guilty because the loss of consciousness could only have been caused by the considerable alcohol ingestion itself.

1005. Oberlandesgericht Hamm [Superior District Appeal Court, Hamm]
URTEIL VOM 3.11.66. [Judgment delivered November 3, 1966].
Blutalkohol (Hamburg), 4(4): 221-223 (10 ref.), 1967.
G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev.
– *CAAAL-0 B-0409.

A case of impaired driving (blood alcohol level 2.1°/oo) is reported. The defense plea was that the intake of drugs (8 “verla-3-dragees” tablets), due to illness, had diminished the defendant’s powers of judgement, and had thus led to excessive alcohol consumption. The conviction was upheld. The court held that every person ingesting alcohol in a state of diminished physical efficiency, of which he must be aware, is responsible for his own state of intoxication. Any claim by the defense of a synergistic alcohol-drug effect, or of excessive alcohol intake due to decreased power of judgement, is therefore irrelevant as long as the accused behaved in a way that could be explained by his blood alcohol level alone. Also reported in Deutsches Autorecht (Munich), 36: 141-142, 1967.

1006. Oberlandesgericht Hamm [Superior District Appeal Court, Hamm]
URTEIL VOM 3.11.66. [Judgment delivered November 3, 1966].
Deutsches Autorecht (Munich), 36: 141-142 (10 ref.), 1967.
G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev.
– *CAAAL-0 B-0410.

A case of impaired driving (blood alcohol level 2.1°/oo) is reported. The defense plea was that the intake of drugs (“verla-3-dragees” tablets), due to illness, had diminished the defendant’s powers of judgement, and had thus led to excessive alcohol consumption. The conviction was upheld. The court held that every person ingesting alcohol in a state of diminished physical efficiency, of which he must be aware, is responsible for his own state of intoxication. Any claim by the defence of a synergistic alcohol-drug effect, or of excessive alcohol intake due to decreased power of judgement, is therefore irrelevant, as long as the accused behaved in a way that could be explained by his blood alcohol level alone. Also reported in Blutalkohol (Hamburg), 4(4): 221-223, 1967.

1007. Oberlandesgericht Hamm [Superior District Appeal Court, Hamm]
URTEIL VOM 24. JUNI 1968. [Judgment delivered June 24, 1968].
Blutalkohol (Hamburg), 7(1): 82-83 (5 ref.), 1970.
G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – CNS –
tranquilizers – *CAAAL-0 B-0545.

A court case is presented in which the defendant, on the day in question, took 4 glasses of cognac and 4 2/10 glasses of beer in the afternoon; then, in the evening at 7 pm, she went with her husband to a tavern where she took 3 glasses of beer and one cognac. The defendant, who was suffering from heart and circulatory problems, had taken 1 valium tablet in the morning and 2 tablets at about 3 pm. She had not read the instructions that, “no alcohol should be taken under the influence of the

drug, since driving ability might be impaired.” At about 12.30 am, the defendant drove her car home, went to the wrong side of the road, and collided with a truck. At the time of the accident, her blood test indicated 2.0 to 2.15‰ alcohol. The court rejected the defendant’s claim that, due to the combined effect of valium and alcohol, she was not responsible for having lost control over the car. It was considered that she should have taken greater precautions against such loss of control.

1008. Oberlandesgericht Oldenburg [Superior District Appeal Court, Oldenburg]
URTEIL VOM 23.4.63. [Judgement delivered April 23, 1963].
Deutsches Autorecht (Munich), 32: 304-306 (8 ref.), 1963.
G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev.
– stimulants – *CAAAL-0 A-0989.

A judgement, rendered by the Oldenburg Superior District Court concerning a physician charged with impaired driving (blood alcohol level 1.8-1.9‰) is reported. The accused admitted drinking 4 glasses of champagne and cognac given him at a party, after ingestion of 2 saridon tablets (which he regularly employed to combat fatigue), and followed by coffee and another 3-4 saridon tablets. He claimed that his intoxication was due to the interaction between alcohol and the drug, the possibility of which he had no prior knowledge. The court declared that, when motor vehicle driving is known to be involved, a physician must be expected to use only those drugs which, on the basis of his own technical knowledge and awareness of the known composition, can be assumed to be safe even when combined with alcohol. The accused was found not guilty, because the medical experts did not agree that the accused could have known the synergistic effect of saridon. Furthermore, it could not be established whether the accused, when he took the last 3-4 saridon tablets, was still able to gauge the effects of the saridon.

1009. Oehme, P., and Richter, H.
GEFÄHRDUNG DURCH REZEPTFREIE SCHLAFMITTEL? [The danger of
non-prescription sleeping pills].
Deutsch. Gesundh. (Berlin), 22: 359-362 (4 ref.), 1967.
G – ES – RS – SEC – general – case hist. – conj. addict. – psychol. perform. – CNS – sed., hypnot.
– *CAAAL-0 B-0968.

The potential hazards of addiction to non-prescriptive drugs are discussed, and a survey of non-prescriptive drug consumption in the German Democratic Republic is presented. 11 case histories of glutethimide abuse are reported, to support the contention that great risks attend free access by the public to non-prescriptive hypnotics. In 1 case, a 40-yr-old married woman with an infantile-demonstrative personality reached a daily intake of up to 3 glasses of brandy, 2 tablets of glutethimide, and up to 60 drops of methylpentynol. 2 yr prior to hospitalization, she was treated for migraine and neurasthenia. 1 yr later, an abnormal jealousy developed, and delusions and hallucinations appeared. When in hospital the patient became delirious, and, a few days later, a hallucinosis with optic and acoustic deceptions developed, lasting 8 weeks. The general physical condition was very poor. From the end of withdrawal treatment until the present, however, the patient has been without complaint.

1010. Oelkers, H. A.
GEWÖHNUNG AN ALKOHOL UND LOKALANAESTHETICA. [Tolerance to alcohol and
local anesthetics].
Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 178:
451-454 (7 ref.), 1935.
G – exp. – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – nerv. syst. –
skel., muscle, skin – anesthetics – *CAAAL-0 A-0990.

The influence of chronic alcohol intake on the anesthetic action of cocaine and other local anesthetics was examined in the cornea of 12 guinea pigs. All animals received, on the first day and then every

fifth day, 2 drops of 0.75% sol of cocaine hydrochloride. The cornea reflex was tested by Regnier's method. In a second experiment, 2 drops of 0.15 and 0.2% cocaine hydrochloride were administered. Chronic alcohol intake in doses of 6, 8, or 12 cc of a 25% alcohol-water sol/kg body wt for 6 weeks was found to be without any effect on the anesthesia in both experiments.

1011. Oelkers, H. A.

LEBERSCHÄDIGUNG UND ALKOHOLABBAU. [Liver damage and alcohol metabolism].

Klin. Wschr. (Berlin), 17(40): 1410-1411 (5 ref.),

1938.

G – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – liver, kidney – anesthetics – unclass. ther. agents – *CAAAL-573-A2 A-0991.

Rabbits were poisoned with arsenic (0.5% sol, dose unstated), phosphorus (3-4 mg/kg po), or chloroform (20-30 min deep anesthesia), thus injuring the liver, and determinations of blood alcohol levels were made after 10 cc 30% alcohol/kg po were given by stomach tube. After arsenic, alcohol oxidation was normal at 48-72 hr after poisoning. Blood alcohol levels were slightly higher 6 hr after alcohol in the phosphorus-treated animals, and, after chloroform, there was nearly always a marked increase in blood alcohol and a deepening of anesthesia. In most of the animals, an increase of urine urobilin and albumin was found after poisoning with arsenic, phosphorus, and chloroform. Often the animals did not seem to be ill, and the gravity of liver poisoning could only be seen from the abnormal blood alcohol curve after alcohol ingestion.

1012. Ogden, E., and Southard, F. D.

THE INFLUENCE OF WINE ON GASTRIC ACIDITY.

Fed. Proc. (Bethesda), 5: 77 (0 ref.),

1946.

E – abst. – exp. comp. – congen. stud. – humans – acute admin. – in vivo – acid-base, blood pH, elect. – G.I. tract – *CAAAL-4338-B1 A-1433.

8 normal male students were studied while fasting, and for 2.5-3 hr after ingestion of soda crackers plus 200 ml of the following test fluids: distilled water, white table wine, 14% ethanol sol, dealcoholized wine, and a cream of tartar sol adjusted to approximate the pH and titratable acidity of the wine. Free and total acidity were titrated on samples withdrawn at 20-min intervals, each test being repeated twice on each subject. After wine, both curves rose higher, reached a peak later, and were more prolonged than after water. After alcohol, the curves resembled the wine curves in intensity and peak, but the acidities tended to rise and fall more rapidly than with wine, and an occasional extremely high acidity occurred. The curves after dealcoholized wine and acid tended to rise little, and to sustain the rise for some time; in some subjects, the rise began early, and, in others, gastric secretion appeared to be completely repressed for 1 hr or more. It is suggested that the effect of the acid or other effects recognizable in the curves after dealcoholized wine may account for the less violent but more sustained stimulation of gastric secretion which occurs after wine, in contrast to alcohol.

1013. Oliveras, E. J.

WHISKEY AS A NEUTRALIZING AGENT TO THE POISON OF THE RATTLESNAKE.

Oglethorpe Medical and Surgical Journal (Savannah), 1(4): 224-228 (0 ref.),

1858-59.

E – general – case hist. – DC (antidotal) – humans – cardiovasc. – *CAAAL-0

A-0992.

Case material is presented concerning a young boy bitten on the lower part of the leg by a rattlesnake. The author found the boy in great pain, with glassy eyes, and a pulse beat of 160, suffering much nausea. Whiskey was administered in 1 oz doses every few min until a sensible impression was made on the pulse. When the last of 8 oz of whiskey was taken, the boy vomited profusely and then fell asleep. Other medication was given for constipation and fever, but, in the author's opinion, the combined sedative and neutralizing effects of the whiskey prevented the patient from dying.

1014. Olszycka, L.

ÉTUDE QUANTITATIVE DES PHÉNOMÈNES DE SYNERGIE: POTENTIALISATION DE L'ACTION HYPNOTIQUE CHEZ LA SOURIS. [Quantitative study of synergistic phenomena: potentiation of action of hypnotics in the mouse].

Académie des Sciences, Comptes Rendus Hebdomadaires des Séances (Paris), 201: 796-797 (6 ref.), 1935.

F – exp. comp. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – elect., water-bal. agents – *CAAAL-0 A-0993.

Mice received a 15% alcoholic glucose sol iv, plus either increasing doses of a bicarbonate sol of 1:200 ethylbutylbarbituric acid, or simultaneous doses of alcohol sol plus the ethylbutylbarbituric acid, in the following combinations: 1) 1.6 mg alcohol/g body wt plus increasing quantities of ethylbutylbarbituric acid varying between 0.04 and 0.065 mg/g body wt, 2) 2.4 mg alcohol/g body wt plus increasing doses of ethylbutylbarbituric acid varying between 0.03 and 0.15 mg/g, and 3) 3.2 mg alcohol/g plus increasing doses of ethylbutylbarbituric acid varying between 0.03 and 0.09 mg/g. For each dose, the coefficient of potentiation (sleeping time of combined hypnotic doses/total sleeping time for the substances administered alone) was calculated. For the most effective instance of potentiation, 3.2 g alcohol/g was combined with 0.03, 0.04, 0.05, 0.06, and 0.065 mg/g ethylbutylbarbituric acid, and the coefficient of potentiation was 14.5, 23.5, 40, 56, and 11.9, respectively. The results show that a combination of inactive doses of hypnotics can produce sleep for 2-78 min, according to the relative proportions used. Combinations of active doses can produce a longer sleeping time than the sum of the sleeping times of the 2 hypnotics administered separately. The potentiation coefficient is most often higher with lower doses of hypnotics.

1015. Olszycka, L.

ÉTUDE QUANTITATIVE DES PHÉNOMÈNES DE SYNERGIE: CONTRIBUTION À L'ÉTUDE DU MÉCANISME DES PHÉNOMÈNES DE POTENTIALISATION DE L'ACTION HYPNOTIQUE CHEZ LE RAT. [Quantitative study of synergistic phenomena: contribution to the study of the mechanism of potentiation of the action of hypnotics in the rat].

Académie des Sciences, Comptes Rendus Hebdomadaires des Séances (Paris), 202: 1107-1109 (1 ref.), 1936.

F – exp. cont. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – CNS – elect., water-bal. agents – *CAAAL-0 A-0994.

Rats received alcohol iv, ethylbutylbarbituric acid iv in a 1:200 carbonated sol, 2.4 mg alcohol in a 30% glucose sol/g plus 0.045 g ethylbutylbarbituric acid, or 3.2 mg alcohol/g plus 0.045 mg ethylbutylbarbituric acid. The coefficient of potentiation (sleeping time of combined doses/total sleeping time of both substances administered separately) was 14.7 for the first combination, and 4.5 for the second. It is concluded that the combination of active doses of the 2 substances produces a sleeping time longer than the sum of the sleeping times of each substance administered individually. There is no difference in alcohol fixation in the blood or brain between control and experimental animals. Potentiation of hypnotic action in the rat is not due to a more complete or facilitated penetration of tissues by alcohol.

1016. Olszycka, L.

CONTRIBUTION À L'ÉTUDE DES PHÉNOMÈNES DE POTENTIALISATION: ACTION DE QUELQUES ASSOCIATIONS D'HYPNOTIQUES. [Contribution to the study of the phenomena of potentiation: action of some combinations of hypnotics].

Dissertation, Faculty of Sciences of the University of Paris, France, 151 pp. (77 ref.), 1938.

F – exp. cont. – exp. comp. – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – cardiovasc. – CNS – G.I. tract – respir. – sed., hypnot. – *CAAAL-0 A-0995.

An extensive study was made of the phenomenon of potentiation, and of the interaction of various substances. A series of experiments were carried out to determine the effect of alcohol (1.6-3.2 mg/g) in interaction with chloral (0.1-0.225 mg/g) and butylethylmalonyl urea (0.03-0.08 mg/g) in rats. The results pointed to a potentiation of the hypnotic action of the drug in interaction with alcohol. The maximum sleeping time achieved was greater in combination with alcohol than when each of the drugs was administered alone at high dosage. The difference in the hypnotic effects between the combined and separate constituents is principally quantitative. The potentiation mechanism is discussed.

1017. Olszycka, L.

SUR QUELQUES ASSOCIATIONS D'HYPNOTIQUES: EXALTATION DE L'ACTION ANESTHÉSIQUE. [Some hypnotic combinations: increase of anesthetic action].

C.R. Soc. Biol. (Paris), 130: 1244-1246 (1 ref.),

1939.

F – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – *CAAAL-762-D2

A-0996.

The combination of 0.07 mg/g ethylisoamylmalonylurea (33% of the toxic dose) and 2.4 mg/g alcohol (54% of the toxic dose), or of 0.06 mg/g isopropylallylmalonylurea and 2.4 mg/g alcohol, appreciably prolonged the anesthetic effect of alcohol in the rat, whereas barbiturates and alcohol administered independently of each other produced no anesthesia at any time. The max sleeping times achieved with a dose of 3.2 mg/g alcohol plus 0.07 mg/g ethylisoamylmalonylurea or 0.06 mg/g isopropylallylmalonylurea were 152 and 167 min respectively. The prolongation of sleep and the onset of anesthesia can be explained by considering the 2 constituents of each combination as being antagonistic in their secondary toxic effects—the animal supports, in combination, greater doses of each constituent than would be the case if the constituents were administered individually, and, hence, the hypnotic and anesthetic effects are increased. However, if the percentages of the constituents of each combination are expressed in terms of the toxic dosages of each substance if given individually, it can be seen that these percentages are not greater than 100%; the alcohol-ethylisomalonylurea combination, for example, establishes and prolongs itself at a combined dose of less than 100% of the toxic doses of its constituents (87%).

1018. Olszycka, L.

RÉPARTITION DE L'ALCOOL ÉTHYLIQUE DANS LES DIFFÉRENTES PARTIES DU SYSTÈME NERVEUX CENTRAL DU RAT, SOUMIS A L'ACTION DE L'ASSOCIATION ALCOOL-BUTYLÉTHYLMALONYLURÉE. [Distribution of ethanol in the different parts of the CNS of the rat, subject to the combined action of alcohol and butylethylmalonylurea].

C.R. Soc. Biol. (Paris), 133: 368-370 (3 ref.),

1940.

F – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vitro – CNS – metab. proc. – *CAAAL-2468-A2

A-1311.

Tests were performed to find the cause of the synergistic effect of butylethylmalonylurea on alcohol. 2.4 and 3.2 mg/g of alcohol were injected into rats, producing unconscious periods of 2 to 3 min, and of approximately 45 min, respectively. With simultaneous injections of 2.4 or 3.2 mg/g alcohol plus .045 mg/g of butylethylmalonylurea (which alone produced an 11 min unconscious period), sleeping times of 191 and 297 min, respectively, resulted. Rats were killed 10 and 60 min after the drugs were administered, and alcohol concentrations in the telencephalon, diencephalon, corpora quadrigemina, cerebellum, annular protuberance, and spinal cord were determined. No changes in concentration of alcohol in any of these tissues were found in the alcohol or butylethylmalonylurea-alcohol groups. The author concludes that the potentiating effect is due to changes in the sensitivity and metabolism of tissues of the CNS, and not to changes in alcohol concentration.

1019. Olszycka, L.

ANTAGONISME DE LA STRYCHNINE ET DE L'ASSOCIATION ALCOOL ÉTHYLIQUE-BUTYLÉTHYLMALONYLURÉE, CHEZ LE RAT. [Antagonism of strychnine and the combination ethyl alcohol-butylethylmalonylurea in the rat].

C.R. Soc. Biol. (Paris), 133: 422-424 (6 ref.),

1940.

F – exp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – stimulants – *CAAAL-2469-D2 A-0997.

Using 158 rats, the toxic dose of strychnine sulphate was determined in control animals and under the following conditions: after 2.4 mg/g alcohol iv, after 0.06 mg/g butylethylmalonylurea iv, and after alcohol and butylethylmalonylurea in combination. The strychnine was given 8-10 min after the other drugs. Both alcohol and butylethylmalonylurea increased the tolerance of the animals to the toxic action of strychnine. When alcohol and butylethylmalonylurea were given simultaneously, the tolerance of the animals to the strychnine was almost equal to the sum of the tolerances found when each of the drugs was given separately.

1020. Olszycka, L.

SUR LE MÉCANISME DES PHÉNOMÈNES DE POTENTIALISATION. II. ACTION DE QUELQUES ASSOCIATIONS D'HYPNOTIQUES. REALITÉ D'UN PHÉNOMÈNE DE POTENTIALISATION. [Mechanism of potentiation phenomena. II. Action of some hypnotic combinations. The reality of a potentiation phenomenon].

Arch. Int. Pharmacodyn. (Gand), 65(4): 467-493 (20 ref.),

1941.

F – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – absorp., distrib., stor. – cardiovasc. – CNS – G.I. tract – respir. – sed., hypnot. – *CAAAL-0 A-0998.

A study was made of three drug combinations administered to rats and mice: (a) alcohol (1.6-3.2 mg/g)-chloral hydrate (0.10-0.3 mg/g), (b) alcohol (0.8-3.2 mg/g)-butylethylmalonylurea (0.03-0.1 mg/g), and (c) chloral hydrate (0.1-0.3 mg/g)-butylethylmalonylurea (0.03-0.06 mg/g). The results indicate that the effects of the combination in (a) are comparable to those obtained with chloral hydrate alone. The combination in (b) produced a marked increase in oxygen consumption and in carbonic gas production, an effect not observed when testing these substances individually. Respiratory changes were observed, and the animals succumbed to a prolonged hypnotic state. The results obtained in (c) were similar to those obtained with barbiturates alone. It is concluded that the potentiation observed is not due to a stronger general penetration of the combined substances, but it could be due to a better elective penetration into the sensory cells of the encephalon.

1021. Olszycka, L.

SUR LE MÉCANISME DES PHÉNOMÈNES DE POTENTIALISATION. III. ACTION DE QUELQUES ASSOCIATIONS D'HYPNOTIQUES. PÉNÉTRATION DES HYPNOTIQUES DANS LES CELLULES SENSIBLES. [Mechanism of potentiation phenomena. III. Action of some hypnotic combinations. Penetration of perceptible cells by hypnotics].

Arch. Int. Pharmacodyn. (Gand), 66(1): 91-112 (20 ref.),

1941.

F – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – blood lev. – other drug lev. – absorp., distrib., stor. – CNS – sed., hypnot. – *CAAAL-0 A-0999.

A study was made on the distribution of hypnotic combinations of: (a) alcohol (2.4 mg/g)-chloral hydrate (0.225-0.2 mg/g), (b) alcohol (2.4 mg/g)-butylethylmalonylurea (0.045 mg/g), and (c) chloral hydrate (0.255 mg/g)-butylethylmalonylurea (0.045 mg/g), in the blood and cerebral tissue of the rat. The results showed that, in (a), the distribution of the 2 substances in the blood and cerebral tissue was the same as when each of these substances was administered individually. The fixation of alcohol and chloral was not influenced by the presence of barbiturates. In the combination alcohol-butylethylmalonylurea, the distribution in the blood and different parts of the CNS was qualitatively and

quantitatively the same as in the animals receiving alcohol alone. It is concluded that the examination of the elective penetration of the hypnotics into the encephalon does not explain the mechanism of potentiation observed with these drug combinations.

1022. Orahovats, P. D., Lehman, E. G., and Chapin, E. W.
POTENTIATING EFFECTS OF QUININE. I. ANALGESICS AND HYPNOTICS.
 Arch. Int. Pharmacodyn. (Gand), 110(2-3): 245-258 (21 ref.), 1957.
 E – SEC – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – CNS – anti-infectants –
 *CAAAL-8374-D2 A-1000.

The effect of quinine, alone or in combination with various other drugs, was studied. 6 rabbits received 2 cc 30% ethanol iv; the average sleeping time was 2.5 min. 3 rabbits were given 2 cc 30% ethanol iv plus 20 mg/kg quinine hydrochloride iv, and the average sleeping time was 10.0 min. The enhancement of the action of a number of chemically unrelated drugs by quinine makes it unlikely that this effect is due to a specific chemical potentiation; it would appear to alter, in some way, the response of the organism to the drugs. No explanation can be offered at the present time, concerning the observation that quinine pretreatment, despite its potentiating effect, does not increase the toxicity of analgesics and hypnotics.

1023. Orenstein, L. L., Bowman, K. M., Kagan, J. R., and Goldfarb, W.
USE OF METRAZOL IN THE TREATMENT OF ACUTE ALCOHOLISM.
 Amer. J. Psychiat. (Hanover, N.H.), 96: 589-594 (5 ref.), 1939.
 E – general – case hist. – DC (antidotal) – drug-dep. humans – blood lev. – CNS – respir. – stimulants
 – *CAAAL-1231-N13 A-1001.

50 alcoholic patients, who could be characterized as either acutely disturbed, offering resistance and violence, or in deep coma or coma complicated by respiratory failure, were given 5 cc metrazol (10% sol) iv immediately upon admission. The improvement in the clinical state was marked. The comatose group was aroused, and the excited group was sedated. These improvements were not due to any changes of the concentration of alcohol in the blood. It is suggested that this apparent biphasic effect of metrazol is due to a direct stimulation of the narcotized cerebral cortex. In the mildly narcotized or agitated group, the improvement is ascribed to a stimulation of the depressed inhibitory centers. Improvement in comatose cases is considered to be due to a general stimulation of the CNS.

1024. Osterhaus, E., and Johannsmeier, K.
UNTERSUCHUNGEN ÜBER DEN EINFLUSS VON JATRONEURAL (TRIFLUOPERAZINDIHYDROCHLORID) AUF DIE RESORPTION VON ALKOHOL UND DEN BLUTALKOHOLABBAU. [The influence of jatroneural (trifluoperazine dihydrochloride) on the absorption of alcohol and on the decrease of the blood alcohol level].
 Blutalkohol (Hamburg), 2: 367-373 (1 ref.), 1964.
 G – exp. cont. – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – acute admin.
 – in vivo – blood lev. – tranquilizers – *CAAAL-0 A-1002.

A court case is described in detail. The defendant drove erratically, and had a blood alcohol level of 2.2°/oo 35 min after arrest. He claimed that he took 4 tablets of jatroneural (2.36 mg trifluoperazine hydrochloride), and could not recall that he had been driving. He was convicted of impaired driving. In an experiment, 6 healthy humans received 1 jatroneural tablet at 8:00 pm, 8:00 am, noon, and 1:30 pm. From 1:30 pm to 3:00 pm, 3 subjects received 200 cc, and the 3 others 250 cc of 43% whiskey. 8 blood samples were taken between 1:30 pm and 6:45 pm, and performance tests administered. A few days previously, performance had been influenced by the drug alone, but performance in the present test was more impaired by the drug-alcohol combination.

1025. Osterhaus, E.

FORENSISCHE BEDEUTUNG VON MEDIKAMENTEN IM STRASSENVERKEHR.

[Forensic importance of drugs in traffic].

Blutalkohol (Hamburg), 2: 395-414 (0 ref.),

1964.

G – exp. – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – acute admin. – in vivo – blood lev. – other drug lev. – CNS – barbiturates – sed., hypnot. – *CAAAL-0

A-1003.

The forensic implications of intake of drugs, alone and in combination with alcohol, are discussed in detail. In an experiment, 8 healthy humans received a therapeutic dose of medinal, and were then allowed to drink what they considered their normal alcohol intake. After 2 hr, 1 subject became unconscious and 5 others showed severe symptoms of intoxication and physical breakdown. These 6 subjects had blood alcohol levels of about 1°/oo, and the other 2 about 0.6°/oo. The latter two did not show much impairment. It is concluded that, in a healthy subject, barbiturate-alcohol synergism can become very dangerous at blood alcohol levels of 1°/oo and more. The reaction can come suddenly and unexpectedly, depending on the time-effect relation of the drug.

1026. Osterhaus, E.

BEGUTACHTUNG BEI SICHERER UND FRÄGLICHER

MEDIKAMENT-EINWIRKUNG. [Certification of drug activity in definite and dubious cases].

Arzneimittelforschung (Aulendorf), 14(8): 923-929 (13 ref.),

1964.

G – ES – exp. – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – acute admin. – in vivo – blood lev. – other drug lev. – CNS – barbiturates – *CAAAL-0

A-0045.

8 students were given 500 mg barbital, and, 2 hr later, were allowed to drink as much beer or gin as they wished. The alcohol intake was between 60 and 130 g, and the blood alcohol levels were between 0.6 and 1.6°/oo. 6 subjects showed severe intoxication—1 became unconscious, 1 almost fell down the stairs, and 1 fell into a long, toxic sleep. The 2 subjects with 0.6°/oo blood alcohol showed little impairment. The critical blood level seems to be around 1°/oo for healthy subjects, but is lower for sick people or for those who receive higher barbital doses. For forensic purposes, it is concluded that, in a forensically significant synergism, the altered toxic condition will be unmistakable in the clinical picture and behaviour of the accused person.

1027. Osterhaus, E.

UNTERSUCHUNGEN ÜBER DAS VERHALTEN NACH EINNAHME VON

NATRIUMBARBITAL, NACHTRÄGLICHER ALKOHOLZUFUHR UND

AUSSCHIEDUNG DER ZUGEFÜHRTE BARBITURSÄURE. [Studies of effects of sodium barbital, subsequent alcohol consumption and of elimination of barbituric acid].

Med. Welt (Stuttgart), 44: 2363-2368 (2 ref.),

1964.

G – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – other drug lev. – CNS – G.I. tract – barbiturates – *CAAAL-11395-D1

A-1004.

7 male human subjects and 1 female were tested for reactions to light and sound, body swaying, and other neurological functions, under the following conditions: after 500 mg sodium barbital, after individually selected amounts of alcohol, and after the same amounts of alcohol given 2-4 and 12 hr after the sodium barbital. Performance was only slightly modified when alcohol was given alone or 12 hr after the barbiturate. When alcohol was drunk 2 hr after the drug, 6 subjects with blood alcohol concentrations of 1.15-1.56°/oo showed severe reactions, including collapse, apathy, and vomiting. The other 2 subjects, with blood alcohol concentrations of about 0.6°/oo, were unaffected. It is concluded that, when alcohol is given during the rise in the effect of soporifics, or when the effects have reached their max, a severe poisoning may suddenly and unexpectedly occur, even though the blood alcohol concentration may be relatively low.

1028. Osterhaus, E.
**WISSENSCHAFTLICHE GRUNDLAGEN UND ERFAHRUNGEN BEI
GLEICHZEITIGER EINWIRKUNG VON MEDIKAMENTEN UND ALKOHOL.** [Scientific
principles and experiences in the simultaneous effects of drugs and alcohol].
Medizinische Sachverständige (Berlin), 60(4): 83-89 (12 ref.), 1964.
G – ES – FS – exp. – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – blood
lev. – CNS – analg., antipyret. – barbiturates – *CAAAL-0 A-1005.

The author discusses the theoretical considerations, clinical observations, experiences of physicians, and experimental investigations which compose the scientific foundation for assessing an impaired-driving offender after combined alcohol-drug intake. In an original experiment, 8 subjects received 50 mg sodium barbital 2 hr before alcohol intake; they were then allowed to drink as much during the following 2 hr as they usually drank. 6 subjects with a 1.0°/oo blood alcohol level showed severe toxic symptoms and complete physical breakdown. 2 subjects with 0.6°/oo blood alcohol showed only normal effects of alcohol. It was concluded that considerable synergism occurred. More experiments led to the same conclusion. It is considered that additive drug-alcohol interaction caused sedation, not excitation. If the addition is not considerable, the subject remains in control of his faculties after a moderate alcohol dosage. A severe alcohol-drug synergism means mortal danger, and the state of intoxication excludes further alcohol intake.

1029. Osterhaus, E.
**DIE GEFÄHRDUNG ALKOHOLISierter PERSONEN DURCH SEDIERENDE
MEDIKAMENTE.** [The danger of administering sedatives to alcoholized persons].
Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 57(1-2): 249-253 (4 ref.), 1966.
G – ES – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – cardiovasc. – CNS
– respir. – analg., antipyret. – sed., hypnot. – *CAAAL-12592-N11 B-0411.

2 fatal cases of alcohol-drug poisoning are reported. The men, alcohol-intoxicated and hyperexcited, were injected with 1 cc of scophedal im. Both fell asleep quickly, but were found dead a few hr later. The histological findings are given. The author considers that any treatment of conditions of excitement caused by alcohol should never be effected in the home of the patient or in police custody, since the danger exists that an immediate respiration paralysis and a circulatory collapse may occur. Even the application of sedatives at relatively low blood alcohol levels may precipitate a drug-alcohol synergism. A continuous medical control over treatment of conditions of excitement caused by alcohol must be ensured.

1030. Ott, I.
**RATTLESNAKE VIRUS: ITS RELATIONS TO ALCOHOL, AMMONIA, AND
DIGITALIS.**
Archives of Medicine (New York), 7: 134-141 (0 ref.), 1882.
E – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – cardiovasc. – respir. –
cardiovasc. agents – *CAAAL-0 A-1006.

Experiments were carried out to determine the effect of alcohol, ammonia, and digitalis in rabbits subjected to rattlesnake bite. The pulse and blood pressure of the animals were measured after the bite, and the following drugs were administered (1 drug/rabbit): 1/2 gutta of ammonia in 1.5 cc water, repeated 3 times; 10 minims of alcohol in 8 cc water; and 6 and 4 cc of digitalis. All 3 drugs temporarily increased the arterial tension which had been lowered by the venom. Ammonia and alcohol increased the pulse rate, whereas digitalis lowered the rate. All of the drugs stimulated the circulation, but the excessive stimulation totally and rapidly exhausted the cardiac irritability. When alcohol and digitalis were combined, the results were about the same as with digitalis alone.

1031. Otto, B. S.

ÜBER TOXISCHE WIRKUNGEN DES ISONIKOTINSÄUREHYDRAZIDS (SUICID MIT 15 G INH). [The toxic effects of isonicotinic acid hydrazide (suicide with 15 g INH)].

Z. Ges. Inn. Med. (Leipzig), 9(21): 1089-1094 (107 ref.),

1954.

G – SEC – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – absorp., distrib., stor. – CNS – anti-infectants – *CAAAL-0

A-1007.

A review of the literature on isonicotinic acid hydrazide (INH) intoxication is followed by a discussion of basic problems in chemotherapy, and of the most common side effects of INH in normal doses. 1 case of suicide is presented. The 19 yr-old patient of a tuberculosis sanitarium drank, for 6 hr, 6 glasses of beer and 1/2 l 35% alcohol. After returning to the clinic, he took 15 g INH. Although treatment started a few min after ingestion of the drug, the patient suffered 23 severe convulsive fits, and died after 16 hr. It is concluded that alcohol increases the toxic affect of INH by increasing absorption. Alcohol must be strictly prohibited for patients receiving INH treatment.

1032. Overholt, B. F.

COMMENT ON ACID, ASPIRIN, ALCOHOL, AND BLEEDING.

Gastroenterology (Baltimore), 56(3): 637-638 (7 ref.),

1969.

E – general – DC (add., infra-add., unspec. incr.) – humans – mammals – acid-base, blood pH, elect. – G.I. tract – *CAAAL-0

B-0412.

The author comments on an article by Davenport, Horace W. (Gastroenterology (Baltimore), 56(3): 439-449, 1969), which reported experimental results showing that, in dogs, ethanol in concentrations as low as 4-5% (w/v) markedly potentiates the damaging effect of acid and aspirin. Several questions are raised, among them, "What is the mechanism by which alcohol potentiates the acid-aspirin injury?" and, "Is there a certain group of patients who are more susceptible to acid-aspirin-alcohol induced injury; e.g. those with blood group A?"

1033. Owens, A. H., Jr., and Marshall, E. K., Jr.

A COMPARISON OF THE METABOLISM OF ETHANOL AND TRICHLOROETHANOL.

Johns Hopkins Hospital, Bulletin (Baltimore), 97: 395-404 (8 ref.),

1955.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – in vitro – blood lev. – liver, kidney – metab. proc. – sed., hypnot. – unclass. ther. agents – *CAAAL-7493-A2

A-1008.

When 0.6 g of ethanol/kg was given to dogs pretreated with disulfiram or cyanamide, the rate of disappearance of alcohol from the blood was not affected. The plasma of dogs which had been pretreated with 0.6 g/kg ethanol, and which were then given 54 mg/kg trichloroethanol iv or po, failed to show any trichloroacetic acid. The repeated administration of ethanol, in an amount sufficient to maintain a plasma level above 20 mg/100 ml, consistently inhibited the oxidation of chloral hydrate (60 mg/kg po) to trichloroacetic acid. The evidence appears to indicate that ethanol and trichloroethanol share common pathways of metabolism.

1034. Paasonen, M. K.

EFFEKTEN VID SAMTIDIG INTAGNING AV ALKOHOL OCH MEDICINER. [The combined action of alcohol and drugs].

Alkoholpolitik (Helsinki), 29(3): 103-106 (20 ref.),

1966.

S – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – absorp., distrib., stor. – cardiovasc. – CNS – metab. proc. – respir. – anti-infectants – barbiturates – enzymes – stimulants – tranquilizers – unclass. ther. agents – *CAAAL-0

B-0413.

The problem of alcohol-drug synergism is discussed with respect to drugs depressing the nervous system, drugs affecting alcohol metabolism, and drugs the absorption of which is increased by alcohol.

Drugs such as caffeine, nikethamide, etc., which inhibit the alcohol effect, are briefly considered. The combined effect of alcohol and drugs, and of drugs in general, is not at all clear yet, and the possibility of unexpected reactions should be borne in mind. When taken at the same time, even small doses of alcohol and a drug may considerably reduce a driver's capability, and lead to fatal consequences.

1035. Pantaleoni, M.

SULLA MAGGIORE TOSSICITÀ CHE L'ALCOOL METILICO RIVELA QUANDO VENGHA MESCOLATO CON ALCOOL ETILICO. [Increased toxicity of methyl alcohol when mixed with ethyl alcohol].

Annali d'Igiene (Rome), 37: 537-541 (12 ref.),

1927.

I – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. – in vivo – CNS – metab. proc. – alcohols – *CAAAL-0 A-1455.

To determine whether the combination of methanol, a common constituent of "sophisticated" liqueurs, with ethanol is responsible for the excessive toxicity of and reported poisonings by such liqueurs, experiments were conducted on cats. The lethal doses of the alcohols were first determined. Following this, it was found that 1300-1400 g cats given 10 g methanol/day did not show any poisoning symptoms for 15-16 days, 2-3 days after which they died. Equivalent ethanol doses produced neither poisoning symptoms or death. 2 animals (3000 and 3500 g) given a sol containing 25% methanol, 25% ethanol, and 50% water po every day or second day (10 g alcohol or less/day) died after a total quantity of only 30 g ethanol + 30 g methanol. It is concluded that the combination of the alcohols is fatal for quantities of alcohol which would easily be tolerated if totally comprised of or even surpassed by only 1 of the alcohols. Thus, with liqueurs containing such combinations, it is not the total amount of alcohol ingested which produces poisoning, because the amount of alcohol is generally insufficient, nor is it the impurities in the beverages; instead, it is a potentiation of the effects of the 2 alcohols. It is suggested that this is due to ethanol being preferentially metabolized in the body, thereby impeding the oxidation and elimination, and increasing the detrimental effects (owing to the demand on the organism for metabolizing methanol at a time when ethanol fully occupies its alcohol-oxidizing capabilities), of methanol.

1036. Paradise, R. R., and Stoelting, V.

CONVERSION OF ACETYL STROPHANTHIDIN-INDUCED VENTRICULAR TACHYCARDIA TO SINUS RHYTHM BY ETHYL ALCOHOL.

Arch. Int. Pharmacodyn. (Gand), 157: 312-321 (18 ref.),

1965.

E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – blood lev. – acid-base, blood pH, elect. – cardiovasc. – barbiturates – *CAAAL-0 B-0414.

10 dogs received an iv infusion of acetyl strophanthidin at a constant rate of 6 µg/kg/min. Within 12 min after the onset of ventricular tachycardia, ethanol was slowly injected iv at dose levels of 200-2000 mg/kg. All dogs receiving 750 mg/kg or more of ethanol, and 1 of the two dogs receiving 500 mg/kg ethanol, were immediately converted to sinus rhythm of varying duration (0.3-10 min). The duration of conversion appeared to be dependent on the dose of alcohol administered. The threshold level of blood alcohol needed for conversion to sinus rhythm was established to be about 240 mg%. Similarities and differences between the effects of ethanol and other anti-arrhythmic drugs are pointed out, and a possible mechanism of action is proposed.

1037. Parker, W. J.

ALCOHOL-DRUG INTERACTIONS.

J. Amer. Pharm. Ass. (Washington), nsv. 10(12): 664-665, 668-673, and 677 (76 ref.),

1970.

E – general – review – cross-tol. – DC (antidotal) – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – absorp., distrib., stor. – cardiovasc. – CNS – liver, kidney – metab. proc. – nerv. syst. – alcohols – amphetamines – analg., antipyret. – antidepressants – anti-infectants

– barbiturates – coagulants – cardiovasc. agents – hormones, hormone antag. – miscellaneous – musculoskel. agents – stimulants – tranquilizers – *CAAAL-0 B-0970.

A pharmacist reviews the literature, and notes that almost half of the most frequently-prescribed drugs in the United States in 1968 had some probability of interaction with alcohol. Caution in dispensing is stressed. The major actions of alcohol on the CNS, the cardiovascular, respiratory, and gastrointestinal systems, and on the liver, adrenal glands, and kidneys, are outlined. The metabolism of alcohol, and the various mechanisms of interaction are discussed. Cross-tolerance between drug and alcohol may occur. Chronic intake of alcohol may increase induction of drug-metabolizing enzymes. Categories of pharmacological interactions are outlined. Detailed tables list a number of drugs, and show possible mechanisms and the clinical significance of their interaction with alcohol.

1038. Pascalis, B.

COMA ETILICO: DIAGNOSI E TERAPIA CON CORAMINA ENDOVENA. [Ethanol coma: diagnosis and therapy with intravenous coramine].

Riforma Medica (Naples), 79: 1057-1061 (55 ref.),

1965.

I – general – case hist. – DC (antidotal) – humans – acute admin. – in vivo – cardiovasc. – CNS – G.I. tract – respir. – stimulants – *CAAAL-0 B-0415.

21 cases of coma are reported. The case reports and experimentation of numerous researchers are evaluated, with regard to the antagonism and therapeutic effect exercised by coramine in acute alcohol intoxication. The doses of coramine employed by some investigators in ethanol coma range from 5 to 100 cc iv. Coramine therapy effected a cure in the majority of cases (75%), and a precise diagnosis derived from the secondary phenomena (successive sneezing) could be formed following iv administration of coramine (in 100% of the cases). Such an indication is of paramount value in the diagnosis of ethanol coma, without resorting to laboratory tests.

1039. Patman, J., Landauer, A. A., and Milner, G.

THE COMBINED EFFECT OF ALCOHOL AND AMITRIPTYLINE ON SKILLS SIMILAR TO MOTOR-CAR DRIVING.

Med. J. Aust. (Sydney), 2: 946-949 (18 ref.),

1969.

E – exp. cont. – DC (unchanged) – mot. vehic. – humans – acute admin. – in vivo – mot. perform. – tranquilizers – *CAAAL-13125-J1 B-0969.

12 men and women, all moderate drinkers, were divided into 4 experimental groups. Groups 1 and 2 received amitriptyline (400 mg/5 days; average of 50 mg/12 hr), and groups 3 and 4 received a placebo. In addition, groups 1 and 3 received on the second test session (fourth day of drug administration) sufficient alcohol (vodka in orange juice) to produce a blood alcohol level of 0.5 g/100 ml, followed by "placebo alcohol" on the third test session; groups 2 and 4 received placebo alcohol on the second test session, and alcohol on the third test session. On each of 3 consecutive mornings, all subjects completed a battery of psychomotor tests: short clerical, dot tracking, pursuit rotor, and simulated driving. In clerical, dot tracking, and pursuit rotor tests, subjects in the placebo drug group performed better than the drug group, whether or not alcohol had been consumed. In the simulation test, the reverse was found, the drug group showing superior performance. While these results were in the expected direction, they were not statistically significant. Analyses of variance of the difference between all means showed a significant performance decrement by alcohol in the pursuit rotor and simulation tests. In no other test did alcohol have a significant effect, and amitriptyline failed to significantly impair performance in any test. No significant interaction effect between amitriptyline and alcohol was noted.

1040. Paulus, W., and Mallach, H. J.

ÜBER ERNÜCHTERUNGSMITTEL FÜR KRAFTFAHRER. II. EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DAS ERNÜCHTERUNGSMITTEL, 'CONTRA' BZW.

,STOP'. [A sobering-up drug for drivers. II. Experimental investigations on the sobering-up drug, 'contra' or 'stop'].

Zentralblatt für Verkehrs-Medizin, Verkehrs-Psychologie und Angrenzende Gebiete (Munich), 1: 92-95 (0 ref.), 1955.

G – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – metab. proc. – *CAAAL-0 A-1009.

Experiments were carried out on the effect of “contra” as a sobriety agent in 12 healthy subjects. The subjects were given beer, wine, and brandy at different time intervals after administration of contra. Blood alcohol analysis was made by the Widmark method, under controlled conditions with alcohol sol of known content. The results (tabulated) indicate that contra had no effect on alcohol metabolism. None of the 12 persons tested showed subjective or objective sobriety. The brief subjective sensory clearing noted in some cases is attributed to the caffeine content in the drug.

1041. Pawan, G. L. S.

AN INVESTIGATION OF FACTORS AFFECTING THE RATE OF METABOLISM OF ALCOHOL (ETHANOL) IN MAN.

Industr. Med. Surg. (Chicago), 39(7): 306 (0 ref.), 1970.

E – abst. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – chronic admin. – in vivo – metab. proc. – stimulants – *CAAAL-0 B-0971.

The effects, on alcohol metabolism (0.5 g/kg) in normal adult human volunteers and some obese subjects, of the following factors were studied: intensive physical exercise (3-mile run or 1000-yard swim), starvation for a period of 7 days (water and adequate vitamins only), 7-day periods on 1000 kcal diets—normal composition, high-fat, high-carbohydrate, high protein plus 50 mg caffeine and/or strong black coffee, various sugars (30 and 60 g po of glucose, galactose, fructose, and sucrose, each sugar being administered on different days), and oral vitamin supplements (10 mg thiamine, 4 mg riboflavin, 150 mg nicotinamide, 20 mg pyridoxine, 600 mg ascorbic acid, 5000 IU retinol, 2 mg calciferol, and 20 mg tocopherols). Each subject served as his own control. Starvation and high-fat diets were found to decrease, while fructose, and, to a lesser extent, sucrose, significantly increased the rate of alcohol metabolism. No other factors had any significant influence.

1042. Pearson, R. G., and Neal, G. L.

OPERATOR PERFORMANCE AS A FUNCTION OF DRUG, HYPOXIA, INDIVIDUAL, AND TASK FACTORS.

Aerospace Med. (St. Paul), 41(2): 154-158 (4 ref.), 1970.

E – exp. cont. – exp. comp. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – CNS – tranquilizers – *CAAAL-0 B-0547.

The effects of alcohol-tranquilizer interaction on the performance of prolonged, complex tasks were studied. 9 male volunteers were tested in groups of 3 on 3 standardized tasks: (1) Model I of the Tracking and Monitoring Task (TMT-I) and associated choice reaction-time equipment; (2) auditory vigilance; and (3) the Welford serial performance, problem-solving apparatus. The 3 variables involved in the 3 x 2 x 2 test design were: tranquilizing drugs, alcohol, and altitude, each with appropriate controls. In the interaction study, 2 doses each of either 400 mg meprobamate or 10 mg librium were administered. 86-proof brandy was given on a body wt basis at the rate of 0.8 cc/lb. The subjects rotated among the tasks for a 3-hr test period. The blood alcohol levels did not differ significantly under drug or altitude conditions. No statistically-significant effect of the drug-alcohol combination on performance was found. The authors discuss possible sources of error in their work.

1043. Pecora, L. J.

PHYSIOLOGIC STUDY OF THE SUMMATING EFFECTS OF ETHYL ALCOHOL AND CARBON MONOXIDE.

Amer. Industr. Hyg. Ass. J. (Detroit), 20(3): 235-240 (22 ref.), 1959.
 E – exp. cont. – DC (unchanged) – mammals – chronic admin. – in vivo – blood lev. – blood comp., sites, lymph – cardiovasc. – CNS – liver, kidney – skel., muscle, skin – *CAAAL-8903-B2
 A-1010.

6 dogs, for a period of 6 hr/day and 5 days/week for 21 weeks, received 15% alcohol in aqueous sol to drink, and were exposed to 0.01% carbon monoxide (CO); immediately prior to CO exposure, 60 ml 15% alcohol was given by stomach tube, and a similar alcohol dose was given 3 hr later to ensure a blood alcohol level of 0.15% during CO exposure. Control groups received alcohol alone or CO alone. No significant summing effects of alcohol and CO were noted. The hemoglobin concentration, electrocardiograms, and body temperature were normal, and the bromsulphalein liver function test was negative. No observable neurological abnormalities were noted. A histological study did not reveal any differences between groups, nor were there any remarkable changes in the animals as a whole.

1044. Pengstritong, K.

INFLUENCE OF ALCOHOL ON THE DISTRIBUTION OF BARBITURATES IN MAMMALIAN TISSUE.

Ph.D. Thesis, University of Illinois, Chicago Professional Colleges, Chicago, Illinois, U.S.A., 71 pp. (133 ref.), 1953.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – absorp., distrib., stor. – CNS – barbiturates – *CAAAL-0
 A-1346.

The potentiating effects of alcohol (3 g/kg po) plus barbital (600 mg/kg sc), phenobarbital (300 mg/kg sc), or pentobarbital (150 mg/kg sc) were studied in 300-400 g albino rats. Induction time, sleeping time, time of death, rate of penetration of barbiturate into the brain, and the pattern of distribution of barbiturate in the body were determined for some of the individual substances and some of the alcohol-barbiturate combinations, and the effects of nalorphine (0.35 mg/kg sc) and oxygen on sleeping time after pentobarbital (25 mg/kg sc) and pentobarbital-alcohol (3 g/kg po) were studied. A method for determination of barbiturate concentrations in animal tissue is described. It was found that alcohol shortened induction time in animals under barbital or pentobarbital anesthesia, but did not affect phenobarbital induction time. Pentobarbital sleeping time was considerably prolonged by alcohol. Barbiturate time of death was markedly shortened by alcohol. Alcohol did not increase the rate of barbiturate penetration into the brain, nor did it change the pattern of distribution of barbiturate in the body. Nalorphine slightly shortened sleeping time after alcohol plus pentobarbital, but did not affect sleeping time after pentobarbital alone. Oxygen inhalation did not affect barbiturate or alcohol-barbiturate sleeping time. It is concluded that alcohol potentiates the action of barbiturates through its histotoxic action on the CNS.

1045. Perisson, J.

ALCOOLISME: TRAITEMENT DES COMPLICATIONS NERVEUSES PAR LA STRYCHNINE. [Alcoholism: treatment of nervous complications with strychnine].

Un. Med. Canada (Montreal), 72: 317 (0 ref.), 1943.
 F – SEC – general – DC (antidotal) – humans – CNS – stimulants – *CAAAL-3856-N7
 A-1011.

The advantages of strychnine for the treatment of nervous complications of alcoholism are pointed out. For treating delirium tremens, doses of 5-10 mg strychnine are administered sc, for a total daily dosage of 2-5 cg; the dosage is decreased to 15-20 mg/day after 2 or 3 days, maintained at this level for 20 days, then gradually reduced to 1 mg/day. As for chronic alcoholism, the initial dosage is 5 mg for the first day, reaching a total dosage of 15-20 mg in the first 48 hr; thereafter, the dosages for delirium tremens are followed. The beneficial effect of strychnine is due to it being an antidote to alcohol, and because it exercises a functional antagonism to the effect of alcohol on the nervous

system, since it decreases the motor chronaxies of the cortical neurons (which alcohol increases). Strychnine appears, however, to have no effect on demented states of chronic alcoholism.

1046. Peter, H.

ALKOHOL UND SEDATIVA. [Alcohol and sedatives].

Dissertation, Medical Faculty of the University of Berlin, 42 pp. (59 ref.),

1939.

G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo
– blood lev. – CNS – analg., antipyret. – barbiturates – sed., hypnot. – *CAAAL-0 A-1012.

Controlled experiments were carried out on the effects of alcohol and sedatives, alone and in combination in human subjects, on the ability to operate a motor vehicle. Alcohol was administered in a dose of 1 g/kg, and the following sedatives were given: luminal (0.3 g), sedormid (0.75g), cibalgine (0.03 g dial plus 0.22 g dimethylaminophenazone/tablet—dose: 3 tablets), and pyramidon (0.4 g). It was found that none of these substances altered the alcohol level. The combined effect of these sedatives with alcohol was dangerous, because of synergism. The relatively inactive cibalgine induced a genuine narcosis in combination with alcohol. Pyramidon and alcohol increased the alcoholic euphoria (subjective and objective), but did not improve performance. An interaction of the tested barbiturates with alcohol concentrations of about 1°/oo was found to cause clinical symptoms simulating much higher blood alcohol levels than really existed (which would justify the toxic effects observed).

1047. Peter, H.

ALKOHOL UND SEDATIVA. [Alcohol and sedatives].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 31: 113-154 (59 ref.),

1939.

G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo
– blood lev. – CNS – analg., antipyret. – barbiturates – sed., hypnot. – *CAAAL-1267-J1

A-1013.

Controlled experiments were carried out on the effects of alcohol and sedatives, alone and in combination in human subjects, on the ability to operate a motor vehicle. Alcohol was administered in a dose of 1 g/kg, and the following sedatives were given: luminal (0.3 g), sedormid (0.75 g), cibalgine (0.03 g dial plus 0.22 g dimethylaminophenazone/tablet—dose: 3 tablets), and pyramidon (0.4 g). It was found that none of these substances altered the alcohol level. The combined effect of these sedatives with alcohol was dangerous, because of synergism. The relatively inactive cibalgine induced a genuine narcosis in combination with alcohol. Pyramidon and alcohol increased the alcoholic euphoria (subjective and objective), but did not improve performance. An interaction of the tested barbiturates with alcohol concentrations of about 1°/oo was found to cause clinical symptoms simulating much higher blood alcohol levels than really existed (which would justify the toxic effects observed).

1048. Peters, U. H.

CHRONISCHE METHYPRYLON-INTOXIKATION UND IHRE PSYCHOPATHOLOGIE.

[Chronic methyprylone poisoning and its psychopathology].

Arch. Psychiat. Nervenkr. (Berlin), 204: 342-348 (26 ref.),

1963.

G – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – CNS – sed., hypnot. – *CAAAL-0 A-1014.

A 50 yr-old man, who had suffered for 20 yr from spondylitis deformans, took 1 g methyprylone together with 1 bottle of beer 2 times/day for 8 months. On 1 occasion, while in a doped state, he took more tablets than usual, and was found unconscious. He was admitted to hospital, and regained consciousness after 5 hr. Psychopathological symptoms included an over-clarity of observation and perception; thinking was facilitated, arithmetic problems were correctly solved at high speed, and movements were very quick but controlled. The patient was euphoric. After 36 hr, he had hallucinations, and thinking became incoherent. On the evening of the fourth day, the psychosis ended with

a long, deep sleep. Complete recovery took several weeks. Reference is made to observations of other authors, showing that methypylone, taken simultaneously with alcohol, has a synergistic effect. Furthermore, the author suspects that methypylone, taken with beer or other alcoholic beverages, can lead to addiction.

1049. Peters, U. H.

KRIMINOLOGISCHE BEDEUTUNG EINER KOMBINATION VON ALKOHOL UND SCHLAFMITTEL. [Criminological significance of a combination of alcohol and hypnotics].

Kriminalistik (Stuttgart), 20(9): 455-456 (11 ref.),

1966.

G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – absorp., distrib., stor. –

CNS – G.I. tract – sed., hypnot. – *CAAAL-0

B-1017.

A relatively recent practice in West Germany, that of persons surreptitiously adding noludar to alcoholic beverages for criminal purposes, is discussed. The advantages of the drug for rendering a victim helpless are pointed out—the drug is virtually harmless, fast-acting, effective, and available without prescription. In combination with alcohol, it rapidly brings on a deep, almost unconscious sleep which lasts for several hours. The effect can be achieved either by ingestion of a large amount of alcohol (approximately 2 litres of beer) plus a small quantity of noludar (400-600 mg), or by consumption of a small amount of alcohol (1 bottle of beer), together with a large quantity of noludar (1-2.5 g). It is suggested that the absorption of noludar is enhanced by alcoholic beverages, probably through the action of alcohol on the mucous membrane of the stomach, and may be further increased through the action of the carbon dioxide present in beer and champagne. The author recommends that misuse of noludar be curtailed by making it available only on prescription.

1050. Peters, U. H.

KOMBINATION VON NOLUDAR UND ALKOHOL: SUCHT UND EINMALIGE VERGIFTUNGEN. [Combination of noludar and alcohol: addiction and poisoning following first use].

Med. Klin. (Munich), 61: 1455-1458 (36 ref.),

1966.

G – general – case hist. – conj. addict. – DC (add., infra-add., unspec. incr.) – humans – absorp., distrib., stor. – CNS – indust. intox. – sed., hypnot. – *CAAAL-0

B-0416.

The literature on the combined intake of noludar (methypylone) and alcohol is reviewed. Reported is the case of a 50 yr-old man who, after discovering that beer considerably increases the effect of the drug, took 1 g noludar plus 1 bottle of beer 2 times/day for several months. On 1 occasion, he took an excessive dose of noludar, and was found unconscious. He was admitted to hospital, and recovered after a few weeks. A female student became addicted to noludar (0.4 g or more) and alcohol, the combination giving her a feeling of euphoria. She had previously been found unconscious several times, and, on 1 occasion, she was admitted to hospital in a coma-like state after ingestion of 2.6 g noludar and 2 glasses of beer. It is hypothesized that not only does the alcohol enhance the noludar effect, but the carbon dioxide in the beer also increases the absorption of the drug. It is pointed out that Hoffer, A. (Canad. Med. Ass. J. (Toronto), 79(3): 191 and 216, 1958) did not observe any synergistic effect after administration of 100 + 50 mg noludar and 6 1/2 oz of rye whiskey.

1051. Peters, U. H.

KOMBINIERTE WIRKUNG VON NOLUDAR UND ALKOHOL: EXPERIMENTELLE UNTERSUCHUNGEN. [Combined action of noludar and alcohol: experimental investigations].

Med. Klin. (Munich), 62(11): 410-414 (17 ref.),

1967.

G – exp. cont. – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – acute admin. – in vivo – mot. perform. – psychol. perform. – species or sex diff. – CNS – analg., antipyret. – sed., hypnot.

– *CAAAL-12600-J1

B-0417.

Several criminal cases are reported in which a combination of noludar (methypylone) and alcohol was used to "knock-out" victims before robbing them. In an experiment, 20 healthy volunteers (15 male, 5 female) were given noludar on different days in doses from 200 mg to 600 mg, alone or combined with 2-6 bottles of beer (4.10% alcohol, 0.49% CO₂). Different co-ordination and psychological tests were used. Ataxia, disinhibition, talkativeness, impaired speech, and hypnotic effects were observed; euphoria was found in all females, but only in a few males. The effects varied widely in the different subjects, but alcohol-noludar synergism was always observed. The author believes that the effect is additive and not potentiative.

1052. Peters, U. H.

ALKOHOL UND MEDIKAMENTE. [Alcohol and drugs].

Fortschritte der Medizin (Berlin), 85(9): 387-389 (44 ref.),

1967.

G – review – DC (add., infra-add., unspec. incr.) – humans – CNS – anti-infectants – barbiturates – sed., hypnot. – unclass. ther. agents – *CAAAL-0

B-0418.

The literature on the effects of alcohol plus drugs is reviewed. The author cites substances like barbiturates, sedatives, tranquilizers, hypnotics, and pain-killers, as showing an additive or synergistic effect in combination with alcohol. He points out that it is difficult to estimate whether the alcohol effect is reinforced by the particular drug or vice-versa. Among the specific drugs cited for their synergism in combination with alcohol are dolviran, somnin, phenobarbital, veronal, noludar, meprobamate, luminal, and aspirin.

1053. Peterson, D. I., Peterson, J. E., Hardinge, M. G., and Wacker, W. E. C.

EXPERIMENTAL TREATMENT OF ETHYLENE GLYCOL POISONING.

J.A.M.A. (Chicago), 186(10): 955-957 (17 ref.),

1963.

E – exp. cont. – DC (decrease) – mammals – dose resp. – other drug lev. – CNS – metab. proc. – indust. intox. – *CAAAL-10593-D2

A-1015.

The LD₅₀'s of ethylene glycol and ethanol were determined in male rats. 10 squirrel monkeys were then given an estimated lethal dose of ethylene glycol ip (as an 80% sol equal to 3.2 ml/kg of pure ethylene glycol). Half of the monkeys were later given 25% ethanol by orogastric tube, as an initial dose equal to 1.5 ml absolute ethanol/kg 30 min after ethylene glycol, and as subsequent doses of 10 ml/kg ethanol every 4 hr for 48 hr. Urine was collected for 48 hr, and the urinary glycol excretion was measured. 6-12 hr after ethylene glycol, the animals receiving ethylene glycol alone became comatose, whereas the animals receiving alcohol plus ethylene glycol were only mildly sedated and slightly uncoordinated. Ethanol-protected animals excreted 10 times as much ethylene glycol as unprotected animals. Tests on rats gave similar results.

1054. Peterson, D. I., Peterson, J. E., and Hardinge, M. G.

PROTECTION BY ETHANOL AGAINST THE TOXIC EFFECTS OF MONOFLUOROETHANOL AND MONOCHLOROETHANOL.

J. Pharm. Pharmacol. (London), 20(6): 465-468 (12 ref.),

1968.

E – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – CNS – alcohols – indust. intox. – *CAAAL-0

B-0419.

The LD₅₀ values of monochloroethanol and monofluoroethanol were determined in rats. The toxicity of these substances in rats subsequently treated with ethanol (25% v/v in water) was then determined. Similarly-treated rats were given an initial dose of 2 ml/kg of ethanol 15 min after the halogenated ethanol, and then 1.5 ml/kg doses every 4 hr. It was found that ethanol increased the LD₅₀ 20 times for fluoroethanol and 4 times for chloroethanol. In another experiment, ethanol appeared to increase, rather than protect against, the toxic effects of isopropyl, n-propyl, n-butyl, and n-amyl alcohols. Tests on monkeys showed a protective effect of ethanol against monofluoroethanol and monochloroethanol.

1055. Pfeifer, E.

EINFLUSS DER DIURESE AUF DEN ALKOHOLGEHALT DES BLUTES. [Influence of diuresis on the alcohol content of the blood].

In: Kionka, H., ed. *Pharmakologische Beiträge zur Alkoholfrage. III.* Jena: Gustav Fischer, pp. 1-44 (66 ref.), 1927.

G – exp. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – other drug lev. – liver, kidney – antispasmodics – *CAAAL-1479-A1 A-1016.

Experiments were conducted on healthy human subjects. Some of the conclusions drawn, concerning the influence of diuresis on normal blood alcohol, are: after diuresis produced by 100 cc of 40% cognac plus 0.5 g theophylline and 1400 cc water, blood alcohol concentration is markedly lower than after 100 cc of cognac alone; after 100-500 cc water plus 0.5 g theophylline, the normal blood alcohol is decreased or disappears completely; and the influence of diuresis is mainly expressed by the fact that concentration in the 2nd hr is lowered, and therefore the blood alcohol curve does not reach the same peak. Since it may be possible to establish a relation between observable symptoms in the patient and the blood alcohol level, the fact that an intensive diuresis can lower the blood alcohol level deserves attention.

1056. Philip, G. E.

ALCOHOL AND DRUGS.

Brit. Med. J. (London), 1(5635): 54 (0 ref.),

1969.

E – general – DC (add., infra-add., unspec. incr.) – humans – CNS – *CAAAL-0 B-0546.

In a letter to the editor, the author comments on a circular printed by the British Committee on Safety of Drugs, and accompanied by a letter from the Medical Assessor, concerning advice to be given to patients regarding alcohol and drug interaction. He challenges the following statement: "a common-sense rule, when you prescribe any drug affecting the CNS, is to warn the patient not to take alcohol whilst under treatment." Such advice to completely ban alcohol consumption would be simply ignored, the writer feels, since there is a great number of persons taking regular doses of drugs which affect the CNS, and many of them naturally wish to drink alcohol. "To tell them not to do so is anything but common sense and is the sort of advice that will be simply ignored to the detriment of both patient and doctor-patient relationship."

1057. Pickford, L. M.

THE SYNERGISM BETWEEN ALCOHOLS AND OTHER DRUGS.

J. Physiol. (London), 63: 19-27 (16 ref.),

1927.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – other org. – in vitro – cardiovasc. – skel., muscle, skin – alcohols – sed., hypnot. – unclass. ther. agents – *CAAAL-0 A-1017.

The effects of methyl, ethyl, n-butyl, and n-octyl alcohols were determined in the isolated frog heart, both alone and in combination with each other and with sodium cyanide and butyl chloral hydrate. It was found that the combinations of alcohols produce an action which can be interpreted as a simple additive effect, provided that the curvature of the relation between concentration and action is taken into account. Combinations of alcohols with either butyl chloral hydrate or with sodium cyanide do not show simple additive effects, but the effects observed can be explained on the assumption that the alcohol covers a portion of the cell surface, and that the other drug acts on the remainder of the surface.

1058. Piker, P.

SYMPTOMS OF ALCOHOLISM.

Cincinnati Journal of Medicine (Cincinnati), 22: 188-193 (0 ref.),

1941.

E – SEC – DC (antidotal) – humans – blood lev. – CNS – stimulants – *CAAAL-3343-A1

A-1018.

A brief review is given of the symptoms of delirium tremens, Korsakoff's psychosis, hallucinosis, pellagra, etc. Physiological and psychological symptoms are described. Clinical symptoms in human subjects were studied. The symptoms which appeared at an alcohol concentration of 194 mg% disappeared completely after 6 hr, although the blood concentration had risen to 250 mg%; whereas it takes only 10-12 min for the rabbit to compensate for the effect of a certain concentration of alcohol in the blood, 6-8 hr are needed for man to compensate. Patients in a deep coma with a blood concentration of 420 mg% regained consciousness after iv administration of metrazol, while blood concentration remained the same.

1059. Pilcher, J. D.

ALCOHOL AND CAFFEIN: A STUDY OF ANTAGONISM AND SYNERGISM.

J. Pharmacol. Exp. Ther. (Baltimore), 3(3): 267-298 (10 ref.),

1912.

E – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – mot. perform. – psychol. perform. – cardiovasc. – CNS – respir. – stimulants – *CAAAL-0

A-1019.

3 series of experiments were conducted on cats: alcohol alone, caffeine alone, or alcohol plus caffeine. Various quantities of 95% alcohol, diluted to 25%, were given by stomach tube. Caffeine was administered as a 1% aqueous sol in doses of 5-200 mg/kg by stomach tube or sc; when combined with alcohol, caffeine was given simultaneously by stomach tube or sc, after symptoms of alcoholic intoxication had developed. The author defines small doses as 2 cc alcohol and 2-10 mg caffeine, moderate doses as 4 cc alcohol and 15-30 mg caffeine, and large doses as 7.5 and 10 cc alcohol and 60 and 120 mg caffeine. It is concluded that the alcohol narcosis is lessened when small or moderate doses of alcohol are combined with small or moderate doses of caffeine (antagonism by algebraic summation of action); narcosis is intensified when moderate doses of alcohol are combined with large doses of caffeine, or when large doses of alcohol are combined with caffeine in all doses (synergism by reversal of caffeine action). With respect to mortality, the 2 drugs are always synergistic, and the mortality rate is greater than simple summation; death results by combining small doses of alcohol with large caffeine doses, and moderate doses of caffeine with moderate doses of alcohol, but not by combining small caffeine doses with large doses of alcohol—alcohol increases the toxicity of caffeine, whereas caffeine does not increase alcohol toxicity.

1060. Pirkner, F.

BISHERIGE KASUISTIK UND BEMERKUNGEN ÜBER DEN GEBRAUCH VON ALKOHOL IN DER BEHANDLUNG VON KARBOLSÄUREÄTZUNGEN UND VERGIFTUNGEN.

[Present casuistry and remarks on the use of alcohol in the treatment of burns and poisonings by carbolic acid].

Deutsche Praxis (Leipzig), 10: 441-448 (5 ref.),

1901.

G – general – case hist. – DC (antidotal) – humans – skel., muscle, skin – anesthetics – anticonvulsants – *CAAAL-0

A-1020.

Several cases (1 fatal) of accidental carbolic acid poisoning are reported. The author also relates his own experiences and experiments, which proved that the internal and external effects of carbolic acid can best be treated with alcohol. Alcohol, when ingested before the carbolic acid is taken, has a prophylactic effect. In all cases of carbolic acid poisoning, alcohol treatment should be given preference, and the following measures taken: 1 or more applications of alcohol, lavage of stomach, application of soluble sulphates, and the usual stimulating methods. In desperate cases, venesection and infusion of saline is recommended.

1061. Pirkner, F.

ON THE USE OF ALCOHOL IN TREATMENT OF CARBOLIC ACID BURNS AND POISONING.

American Medicine (Philadelphia), 1: 358-359 (6 ref.), 1901.
 E – abst. – general – case hist. – DC (antidotal) – humans – absorp., distrib., stor. – skel., muscle, skin – anesthetics – anticonvulsants – *CAAAL-0 A-1021.

This is a long abstract of a paper presented in 1901. A case concerning a 3 1/2 yr-old child who had swallowed at least 2 drams of carbolic acid is reported. 3 1/2 oz of absolute alcohol was administered, followed by 1/60 grain strychnine twice hypodermically, and a hot bath (100°F). The alcohol brought about a regular stupor, the pulse rate improved, and in 7 hr the child was conscious. The patient died 15 hr after poison ingestion, and the author attributes this to the late start (1/2 hr) of treatment. It is advised that the alcohol dose should not be repeated if it has no chance to exert antidotal action, due to a lack of a corresponding quantity of chemically unchanged carbolic acid in circulation; an excess of alcohol may be hazardous. The full text of the paper is published, in German, in *Deutsche Praxis* (Leipzig), 10: 441-448, 1901.

1062. Pletscher, A., Besendorf, H., Steiner, F. A., and Gey, K. F.
THE EFFECT OF 2-HYDROXY-BENZOQUINOLIZINES ON CEREBRAL 5-HYDROXYTRYPTAMINE, SPONTANEOUS LOCOMOTOR ACTIVITY, AND ETHANOL HYPNOSIS IN MICE.
Med. Pharmacol. Exp. (Basel), 7: 15-20 (7 ref.), 1962.
 E – FS – GS – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – other drug lev. – mot. perform. – CNS – metab. proc. – tranquilizers – *CAAAL-0 A-1434.

Mice, fasted for 16 hr, were injected with 1 of 7 2-hydroxy-hexahydrobenzo[a]quinolizines (2-HBQ). At various intervals, 5-hydroxytryptamine (5-HT) was measured in brain homogenates, and the degree of sedation, as indicated by spontaneous locomotor activity, was assessed by a photographic method. The sleeping time of mice pretreated with a 2-HBQ was measured after injection of 4 g/kg ethanol (10% sol in 0.9% saline) ip. All 2-HBQ's decreased locomotor activity, and potentiated the effect of the subhypnotic ethanol dose. No strict correlation was established between diminution of cerebral 5-HT and the reduction of locomotor activity or the enhancement of ethanol hypnosis. 4 of the drugs lowered 5-HT in the brain, and 3 had no effect, thus suggesting that the 2-HBQ's may cause sedation by 2 different mechanisms. It seems unlikely that 5-HT release is a major factor in ethanol potentiation. Some derivatives diminished 5-HT, but only slightly increased the hypnotic effect of ethanol, while others potentiated ethanol without influencing the 5-HT level.

1063. Plochmann, E.
DIE BEEINFLUSSUNG DER WIRKUNGEN VON ARZNEIMITTELN UND GIFTEN DURCH ALKOHOL. [The influencing by alcohol of the effects of drugs and poisons].
 Dissertation, Medical Faculty of the University of Würzburg, Germany, 30 pp. (28 ref.), 1920.
 G – review – DC (antidotal) – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – mammals – anesthetics – diagnost. agents – miscellaneous – sed., hypnot. – stimulants – unclass. ther. agents – *CAAAL-0 A-1022.

The literature on the combined effects of alcohol and drugs or chemical substances is reviewed in detail, and the symptoms of alcohol poisoning are described. The literature on chloroform, bromides, chloral hydrate, strychnine, caffeine, nicotine, cocaine, salicylates, metallic poisons, benzol, aniline, and calcium cyanide, in combination with alcohol, is discussed in detail. No original work is reported.

1064. Podgainy, H., and Bressler, R.
BIOCHEMICAL BASIS OF THE SULFONYLUREA-INDUCED ANTABUSE SYNDROME.
Diabetes (New York), 17(11): 679-683 (28 ref.), 1968.
 E – exp. cont. – DC (supra-add. incr.) – mammals – acute admin. – in vitro – liver, kidney – metab. proc. – elect., water-bal. agents – unclass. ther. agents – *CAAAL-13375-B2 B-0972.

Metabolic changes thought to be involved in producing the antabuse syndrome were studied in vitro. The effects of chlorpropamide and tolbutamide on acetaldehyde, and the changes in amounts of serotonin metabolites under various conditions, were measured in liver homogenates from male guinea pigs. Both chlorpropamide and tolbutamide non-competitively inhibited oxidation of liver aldehyde dehydrogenase (LAD). Chlorpropamide appeared to be the more potent inhibitor of the enzyme. It caused a decrease in 5-hydroxyindoleacetic acid (the major in vivo serotonin metabolite) of 12-19%, and increased production of neutrals by 21-41%. The addition of ethanol to the reaction mixture stimulated the effect of chlorpropamide on serotonin metabolism, and the combined effects were found to be more than additive. It is concluded that, whether the antabuse syndrome results from an accumulation of acetaldehyde due to a block in ethanol metabolism, or whether it is the result of an alteration in serotonin metabolism, the non-competitive inhibition of LAD by the sulfonylureas, coupled with the actions of ethanol, may be considered as a potential cause.

1065. Pogátsa, G., Káldor, A., Bellus, E., and Somogyi, E.
 ZUR WIRKUNG VON INSULIN, CARBUTAMID UND 2-DESOXY-D-GLUCOSE BEI
 DER AKUTEN ÄTHYLALKOHOLVERGIFTUNG DER RATTE. [The effect of insulin,
 carbutamide, and 2-desoxy-D-glucose in acute ethyl alcohol poisoning in the rat].
 Klin. Wschr. (Berlin), 43(11): 611-613 (11 ref.), 1965.
 G – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute
 admin. – in vivo – blood lev. – metab. proc. – hormones, hormone antag. – *CAAAL-11526-A2
 B-0973.

The connection between the metabolism of alcohol and carbohydrate metabolism was investigated by studying, in 2 series of experiments on rats, the effects on alcohol metabolism of substances influencing the metabolism of carbohydrates. In the first series, groups of white rats (250-400 g) received 1 of the following administrations: 2 ml/kg saline sol ip, 2 ml/kg 25% alcohol sol ip, alcohol (same dose) + 0.25 IU insulin/kg iv, alcohol (same dose) + 2 g/kg 40% glucose sol + insulin (same dose) iv, alcohol (same dose) + 100 mg 10% carbutamide sol ip, or alcohol (same dose) + 200 mg/kg 10% 2-desoxy-D-glucose sol. Before injections, and for 3 hr afterward, alcohol, sugar, and pyruvic acid levels were determined in blood. In the second series, rats received the above alcohol dose + the above iv doses of either insulin or carbutamide, and blood sugar levels were determined. Neither alcohol nor pyruvic acid levels were affected by the drugs. Blood sugar levels increased slightly after alcohol. Carbutamide and 2-desoxy-D-glucose tended to retard alcohol elimination. Alcohol inhibited carbutamide-induced hypoglycemia. It is concluded that there is no close correlation between alcohol and carbohydrate metabolism, and that the administration of substances affecting the latter in cases of acute alcohol intoxication is not justified.

1066. Pohl, W.
 KOMBINIERT ALKOHOL-MEDIKAMENTEN-WIRKUNG UND IHRE BEDEUTUNG BEI
 VERKEHRSUNFÄLLEN. [Combined effects of alcohol and other drugs, and their importance in
 traffic accidents].
 Dissertation, Medical Faculty of the University of Munich, West Germany, 51 pp. (38 ref.), 1964.
 G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev.
 – CNS – amphetamines – analg., antipyret. – anti-infectants – barbiturates – coagulants – cardiovasc.
 agents – tranquilizers – *CAAAL-0 A-1023.

The interaction of alcohol and drugs, the symptoms observable in subjects, and the effect of such combinations on driving ability are discussed. The chief characteristics of different psychoactive drug groups are outlined. The main content of the dissertation is a review of forensic aspects. The legal basis of traffic law is determined, and a number of court cases involving alcohol-drug synergism and potentiation are presented. It is pointed out that severe impairment can occur at low blood alcohol

levels, and also very suddenly. Finally, it is questioned whether drugs fall under the legal definition of "other intoxicating substances". No original work is reported.

1067. Pöldinger, W.

PSYCHOPHARMAKA UND ALKOHOL UNTER BESONDERER BERÜCKSICHTIGUNG VERKEHRSMEDIZINISCHER PROBLEME. [Psychopharmacological drugs and alcohol with special consideration of driving problems].

Praxis (Berne), 53(27): 926-934 (50 ref.),

1964.

G – ES – FS – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – psychot. humans – drug-dep. humans – blood lev. – other drug lev. – psychol. perform. – species or sex diff. – CNS – senses – anti-infectants – barbiturates – tranquilizers – unclass. ther. agents – *CAAAL-0 A-1024.

Reviewed is the literature dealing with the problem of the medical use of psychotropic drugs, and their alcohol-potentiating activity in relation to traffic safety. The effects on the total personality are discussed, and the dangers of careless drug use by traffic participants are emphasized. Hypnotics, composite sedatives, and pain-relievers containing such compounds, potentiate the effects of even small quantities of alcohol. Since they are excreted slowly, their alcohol-potentiating effect may persist into the next day after a night-time dose. Therefore, after an accident, not only alcohol intake but also drug administration in the 24 hr preceding the accident should be checked.

1068. Pöldinger, W., and Sutter, W.

DIE BEEINFLUSSUNG DER VERKEHRSTÜCHTIGKEIT DURCH PSYCHOTROPE PHARMAKA. [The influence of psychotropic drugs on driving ability].

Pharmakopsychiatrie Neuro-Psychopharmakologie (Stuttgart), 1(3): 223-231 (87 ref.),

1968.

G – SEC – exp. comp. – review – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – drug-dep. humans – CNS – barbiturates – sed., hypnot. – tranquilizers – *CAAAL-0 B-0420.

A literature review is given on the effect of psychotropic drugs in vehicular traffic and of interactions of these drugs with alcohol. Reference is made to field tests showing a significant synergistic effect in interactions between alcohol (0.8°/oo) and the following drugs: chlordiazepoxide (20 mg), meprobamate (800 mg), phenobarbital (200 mg), and methyprylone (200 mg). Chronic application of these drugs results in habituation effects, with diminished danger of driving impairment and synergism with alcohol. It is estimated that up to 16% of traffic accidents are due to alcohol and drugs.

1069. Pons, C. A., and Custer, R. P.

ACUTE ETHYLENE GLYCOL POISONING: A CLINICO-PATHOLOGIC REPORT OF EIGHTEEN FATAL CASES.

Amer. J. Med. Sci. (Philadelphia), 211: 544-552 (10 ref.),

1946.

E – SEC – stat. surv. – DC (decrease) – humans – CNS – liver, kidney – indust. intox. – *CAAAL-4877-E5 A-1025.

18 fatal cases of acute ethylene glycol poisoning are presented. Mention is made of the fact that death occurred 22 to 44 hr after ingestion of the fluid, except in 1 instance in which a man drank several glasses of vodka; the onset of his symptoms was delayed for 2 days, and he lived 3 days after that. Lesions of the central nervous system were found to be responsible for the deaths. Renal damage severe enough to contribute to the fatal outcome was found in only 1 patient.

1070. Ponsold, A.

ALKOHOL UND MEDIKAMENT. [Alcohol and drugs].

In: Glass, Theo, et al., eds. *Alkohol und Alkoholismus: 27 Internationaler Kongress.* [Alcohol and

alcoholism: 27th International Congress]. Hamm, Westf., West Germany: Deutsche Hauptstelle gegen die Suchtgefahren, pp. 133-136 (0 ref.), 1965.
 G – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – blood lev. – CNS – barbiturates – stimulants – tranquilizers – *CAAAL-0 B-0421.

This is a summary of the known effects of alcohol-psychoactive drug combinations on driving performance. It is noted that barbiturates can lead to sudden sleep or even complete breakdown. Stimulants excite and make one overconfident. Tranquilizers over-excite or make one apathetic. In case of illness, all drugs reduce the already lower-than-normal alcohol tolerance. Many drugs have a long-lasting effect on the CNS even after having been excreted from the body; they have synergistic effects with alcohol although they are no longer detectable. There is a simple rule for suspecting interaction: if a driver acts in all tests in conformity with his established blood alcohol level, interaction can be ruled out for all practical purposes. But, if he acts in a way which only a considerably higher blood alcohol level would justify, there is a strong possibility (although not certainty) of synergistic interaction.

1071. Portmann, G., and Descaux, J.

A PROPOS DE RÉACTIONS PROVOQUÉES CHEZ LES ALCOOLIQUES PAR LES ANESTHÉSIFIQUES DE BASE. [Concerning reactions in alcoholics provoked by basic anesthetics].

J. Med. Bordeaux (Bordeaux), 123: 179-180 (0 ref.), 1944.
 F – abst. – general – DC (add., infra-add., unspec. incr.) – drug-dep. humans – CNS – anesthetics – autonomic agents – *CAAAL-0 A-1026.

2 patients with a history of alcohol addiction were anesthetized by a hypodermic injection of scopodol (Merck) in one person, and a compound corresponding to the Merck product, prepared by the author, in the other. Local anesthesia was also applied—a novocaine sol (1/200 adrenaline) to the first patient, who had a partial maxillary resection, and a cocaine sol (1/10) to the other for an esophagoscopy. The former experienced delirium tremens terminating in exitus, and the latter a state of confusion lasting 12 hr. The authors conclude that alcoholic patients are particularly susceptible to reaction with scopolamine. Further research is needed to elucidate the reaction mechanism.

1072. Powell, S. D.

CARBOLIC ACID IN SURGERY.

Medical Record (New York), 55: 372-373 (0 ref.), 1899.
 E – SEC – general – case hist. – DC (antidotal) – humans – skel., muscle, skin – anesthetics – *CAAAL-0 A-1027.

The author describes the benefits of treating various diseases and injuries with carbolic acid, and of using alcohol as an antidote to the acid. He states that he knows of three cases in which alcohol was successfully used, and estimates that a 25% sol of alcohol is sufficient to counteract the strongest sol of acid possible. Whiskey, brandy, and some wines may suffice as an antidote. One case reported concerned a 1 month-old boy who had been given strong carbolic acid by mistake. The attending physician immediately put a teaspoonful of alcohol down the child's throat. A successful recovery was made, although the parts had been almost black by the time treatment was given. Due to exposure, the child died a week later from pneumonia. The autopsy showed absolutely no damage to the oesophagus or stomach.

1073. Prag, J. J.

THE CHEMICAL AND THE CLINICAL DIAGNOSIS OF "DRIVING UNDER THE INFLUENCE OF ALCOHOL" AND THE USE OF THE CHEMICAL TESTS IN TRAFFIC LAW ENFORCEMENT.

South African Journal of Clinical Science (Capetown), 4(4): 289-325 (34 ref.), 1953.
 E – SEC – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – med.-leg.
 – humans – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – CNS – metab. proc. –
 barbiturates – *CAAAL-6738-A15 A-1028.

The author discusses the absorption, combustion, and excretion of alcohol; alcohol tolerance; clinical diagnosis of intoxication; methods of determining blood alcohol concentrations; and legal aspects of chemical tests for intoxication. 10 experiments related to the above topics were performed. In 1 experiment, the effect of barbiturates on the blood alcohol concentration (BAC) was determined. 3 human subjects received 0.75 g alcohol/kg, followed 1 hr later, in successive tests, by 1 g phenobarbitone, 1 1/2 g seconal sodium, or 1 1/2 g sodium amytal. In controls, the max BAC was reached in 2 hr. When phenobarbital was given, the max BAC was also reached in 2 hr, but, after seconal sodium and sodium amytal, it took 3 hr to attain the max BAC. Seconal sodium and sodium amytal thus retard alcohol combustion, and can result in a higher BAC, thus increasing the rate of absorption. In the absence of barbiturates there was no clinical evidence of intoxication. In the presence of 1 g phenobarbitone, there was also no intoxication, but, in the presence of both seconal sodium and sodium amytal, there were clinical signs of intoxication present.

1074. Prag, J. J.

THE CHEMICAL DIAGNOSIS OF ALCOHOLIC INTOXICATION.

S. Afr. J. Med. Sci. (Johannesburg), 18: 141-154 (17 ref.), 1953.
 E – SEC – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin.
 – in vivo – blood lev. – absorp., distrib., stor. – CNS – metab. proc. – *CAAAL-6738-A15
 A-1029.

The difficulties involved in the diagnosis of drunkenness are pointed out. Conditions such as emotional shock, excitement, fatigue, acute fevers, and fumes of carbon monoxide may all simulate drunkenness. Poisoning by drugs (e.g., belladonna, which produces emotional excitement, dilation of the pupils, flushing of the face, dryness of the tongue, and indistinctness of articulation), diabetes, hypoglycemia, tabes dorsalis, hyperthyroidism, and many other chronic diseases, may also lead to an incorrect diagnosis being made. Various experiments on human subjects concerning the combustion, absorption, and elimination of alcohol were made. Among other results, it was found that sugars increase the degree of intoxication, but assist in sobering the individual more quickly, as they increase the rates of combustion and elimination of alcohol.

1075. Preller, A. C. N., and Davie, G.

DIXYRAZINE (ESUCOS) IN DIE BEHANDELING VAN AKUTE ALKOHOLISME.

[Dixyrazine (esucos) in the management of acute alcoholism].

S. Afr. Med. J. (Capetown), 41(36): 909-910 (0 ref.), 1967.
 A – SEC – DC (add., infra-add., unspec. incr.) – drug-dep. humans – CNS – barbiturates – sed.,
 hypnot. – *CAAAL-0 B-0422.

62 male alcoholics with acute withdrawal symptoms were treated with dixyrazine, a phenothiazine derivative. 26 of the patients were given an initial dose of 70 mg, 1/2 of which was administered iv, and 1/2 given im (simultaneously). This dosage proved insufficient to alleviate the symptoms in some cases. The other 36 patients were given an initial dose of 160 mg dixyrazine, of which 100 mg was administered iv, and 60 mg given im. Very satisfactory results were obtained. Only 1 patient showed extra-pyramidal attacks, and 2 showed hypotensive disturbances. It is noted that the drug potentiates the sedative effect of alcohol already present in the patient, and therefore has a very calming effect without the agitative depression which often occurs after barbiturate administration. Dixyrazine controls the urge to vomit, quickly restores the body equilibrium and the electrolyte and nutritional balance, and reduces anxiety.

1076. Pribilla, O.

KURZNARKOSE UND SCOPHEDAL BEI UNRUHIGEN ALKOHOLIKERN? NACH EINEM FALL AUS DER GUTACHTERPRAXIS. [Short narcosis and scophedal in agitated alcoholics? A case from the practice of a court expert].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 52: 406-423 (28 ref.),

1962.

G – general – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – blood lev. – anesthetics – autocoids – barbiturates – sed., hypnot. – stimulants – tranquilizers – *CAAAL-0 A-1030.

Case material is presented to illustrate medico-legal implications of the treatment of alcohol abuse. Treatment of 1 patient included luminal, megaphen, atosil, and 10 ml baytinal for sedation. Following his release, the patient was under ambulatory treatment, and was treated on readmission to the hospital with iv injections of narcotics and inaktin (6 and 4 ml respectively), followed 1/2 hr later by scophedal injection, and then lobelin. The patient died shortly afterwards. The blood alcohol level was determined at 2.1°/oo. The question of an adverse reaction of inaktin and scophedal with alcohol is raised.

1077. Proctor, C. D., Denefield, B. A., Ashley, L. G., and Potts, J. L.

EXTENSION OF ETHYL ALCOHOL ACTION BY PILOCARPINE.

Brain Res. (Amsterdam), 1(3): 217-220 (10 ref.),

1966.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – CNS – autonomic agents – *CAAAL-12599-D2 B-0423.

Loss of righting reflex was studied using 3 groups of 10 rats each. The first group received 20 mg/kg atropine methylbromide 40 min before, and 5 mg/kg pilocarpine 20 min before, the ip injection of 3 g/kg ethanol. The second group received saline, and the third, 5 mg/kg atropine sulphate and 5 mg pilocarpine before ethanol. The mean sleeping time was increased to 19.5 min in group 1, and to 1.0 min in group 3, as compared to 0 min in group 2 (control). The pilocarpine potentiation of ethanol action is apparently exerted when this cholinergic agent is administered subsequent to administration of atropine methylbromide, but not when it is given following administration of atropine sulfate. The pattern of the influence of these 2 anticholinergic agents on the pilocarpine alternation of ethanol anesthetic action would make it appear that the response to pilocarpine is more related to a CNS action than to a peripheral action of this cholinergic agent.

1078. Prüll, G.

ÜBER DIE WIRKUNG VON WECKMITTELN (COFFEIN UND PERVITIN) AUF DIE PSYCHOPHYSISCHE LEISTUNGS-FÄHIGKEIT NACH ALKOHOLGENUSS. [The effect of stimulants (caffeine and pervitin) on psycho-physical performance after alcohol ingestion].

Dissertation, Medical Faculty of the University of Frankfurt, West Germany, 56 pp. (52 ref.),

1955.

G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (decrease) – humans – acute admin. – in vivo – psychol. perform. – cardiovasc. – CNS – amphetamines – stimulants – *CAAAL-0 A-1031.

Experiments with 5 healthy students were carried out to determine the influence of caffeine and pervitin on mental and physical performance following ingestion of alcohol. The subjects received at different times: 150 cc 38% alcohol, 4.8 g caffeine (4 cups of strong coffee), 800 cc Coca-Cola containing 0.1 g caffeine, 12 mg pervitin, or 1 of these substances together with alcohol. The experiments are described in considerable detail. It was found that the mental performance, impaired by alcohol, was quantitatively improved, but qualitatively adversely affected by the drugs. The already exaggerated physical stress was, under exhaustion of all physical reserves, still increased. This can lead to very dangerous situations, and to cardiovascular breakdown. The use of the foregoing stimulating drugs to restore physical or mental performance after alcohol intake can not be recommended.

1079. Pullar-Strecker, H.
INTRAVENOUS DETOXICATION OF DRUNKENNESS.
Brit. Med. J. (London), 1: 935 (1 ref.), 1953.
E – SEC – general – DC (add., infra-add., unspec. incr.) – humans – CNS – barbiturates – nutritive agents – *CAAAL-6478-N4 A-1032.

This letter is in response to the Nobes (Brit. Med. J., 1: 836, 1953)-Bartley (Brit. Med. J., 1: 163, 1953) correspondence. The author agrees with Nobes that iv barbiturates are dangerous in drunken patients. He suggests a method of detoxifying the drunken patient, consisting of an iv injection of a high dose of multiple-vitamins.

1080. Pullar-Strecker, H.
NOTES ON SUBSTANCES OTHER THAN TED CAUSING DISTASTE,
DISINCLINATION, OR DISLIKE FOR ALCOHOL.
International Journal on Alcohol and Alcoholism (Oxford), 1: 98-102 (76 ref.), 1955.
E – review – DC (sensit.) – *CAAAL-7512-C3 A-1347.

References pertaining to substances other than disulfiram, causing distaste, disinclination, or dislike for alcohol are presented. The literature regarding hydrogen sulphide, tetraethyl lead, alcohol-induced pain in Hodgkin's disease, alcohol-induced flushing in metastizing carcinoid tumors, cyanamide, *Coprinus atramentarius*, animal charcoal, and irgapyrine, is reviewed and discussed.

1081. Pullar-Strecker, H.
ADDENDUM TO: NOTES ON SUBSTANCES OTHER THAN TED CAUSING DISTASTE,
DISINCLINATION OR DISLIKE FOR ALCOHOL.
International Journal on Alcohol and Alcoholism (Oxford), 1: 171 (3 ref.), 1955.
E – review – DC (sensit.) – humans – cardiovasc. – anti-infectants – *CAAAL-7512-C3 A-1348.

In an addendum to a previous review of literature, studies concerning alcohol-induced pain in Hodgkin's disease, isoniazid, and rhodan preparations are reviewed. Both of the latter 2 substances appear to be able to produce intolerance to alcohol.

1082. Pullar-Strecker, H.
ADDENDUM NO. 2 TO: NOTES ON SUBSTANCES OTHER THAN TED CAUSING
DISTASTE, DISINCLINATION OR DISLIKE FOR ALCOHOL.
International Journal on Alcohol and Alcoholism (Oxford), 2: 40-42 (9 ref.), 1957.
E – review – DC (sensit.) – humans – mammals – acute admin. – in vivo – blood lev. – glands – unclass. ther. agents – *CAAAL-7512-C3 A-1312.

4 substances are dealt with in this review of experimentation by other researchers: citrated calcium carbimide (CCC), disulfiram, n-butyraldoxime, and thyroid preparations. CCC is reported to have therapeutic advantages over disulfiram. While it is only effective up to 24 hr, less than disulfiram, it does not have the unpleasant side effects of the latter. 19 patients took 50 to 100 mg of CCC per day for 4 months. None who had previously complained of drowsiness with disulfiram had the same problems with the new drug. Thyroid preparations, when fed to rats as 0.1% of their diet, were effective in creating a distaste for alcohol. Concentrations as low as 0.04 were found to be effective. It was noted that rats, the thyroids of which were removed, developed a preference for alcohol sol over stock sol. N-butyraldoxime was found to produce hypersensitivity to alcohol. This antioxidant was used as an anti-skinning compound in a large printing establishment. 240 men working there suffered flushing and palpitation if they consumed alcohol the same evening after work. It proved to be a classical antabuse-alcohol effect, the solvent raising the acetaldehyde level in the blood to 500 gamma per cent.

1083. Püllen, C.
 ERFAHRUNGEN MIT PERVITIN. [Experiences with pervitin].
 Munchen. Med. Wschr. (Munich), 86(26): 1001-1004 (9 ref.), 1939.
 G – SEC – exp. – DC (decrease) – humans – acute admin. – in vivo – CNS – amphetamines –
 *CAAAL-0 A-1033.
- Some case material is presented, and the author reports his own experimental and clinical experiences. Pervitin was found to be a highly effective psychic stimulant, and showed a marked stimulatory effect on the circulation. It antagonized the unpleasant side effects of morphine therapy, but not the analgesic effect. To establish the effect of alcohol plus pervitin, tests were made on human subjects, the best results being obtained with a dose of 6-9 mg pervitin (2-3 tablets). The drug was sometimes found substantially to elevate tolerance to alcohol, and at other times to have a rapid sobering effect on mildly or severely intoxicated persons.
1084. Pusch, H.
 EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DEN VERLAUF DER
 BLUTALKOHOLKURVE BEI PROTRAHIERTER ALKOHOLAUFNAHME UND BEI
 COFFEINGABE. [Experimental investigations on the effect of protracted alcohol and caffeine
 administration on the progress of the blood-alcohol curve].
 Dissertation, Faculty of Medicine of the University of Göttingen, Germany, 48 pp. (19 ref.), 1938.
 G – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – humans – in vivo – dose resp. –
 blood lev. – psychol. perform. – absorp., distrib., stor. – CNS – metab. proc. – stimulants –
 *CAAAL-0 A-1435.
- A 6-week experiment on 2 human subjects studied the influence of caffeine on the blood alcohol curve (BAC) and the effects of alcohol. Sober days were alternated with test days, on which alcohol, or alcohol followed by caffeine, were given. On test days, a 9-hr test was conducted, in which a total of 116.7 g absolute alcohol was given as beer in 5 doses (1000, 250, 750, 250, and 750 cc, respectively) over a 2 1/2-hr period. Coffee, prepared from 40 g coffee beans plus 400 cc water, was given 25 min, and again 130 min, after the last alcohol dose. Blood samples were taken at 25-min intervals, and performance and other tests were made at 60-min intervals. The BAC and rate of alcohol metabolism were not in any way affected by caffeine. The caffeine did temporarily counteract the subjective and objective effects of alcohol for periods of 15-30 min after the coffee. The subjects felt less fatigued, gait was steadier, speech impairment disappeared, and reaction time decreased (but not to the normal interval). Later on, after the coffee, there was a feeling of pronounced relaxation. Strong diuresis which followed alcohol intake was not affected by caffeine; however, the latter did offset the decreased blood pressure and pulse rate of the former.
1085. Quadland, H. P.
 CARBON TETRACHLORIDE—PART 2 OF A LITERATURE STUDY OF REPORTS OF
 OCCUPATIONAL INJURIES ATTRIBUTED TO VOLATILE SOLVENTS.
 Industr. Med. Surg. (Chicago), 12(12): 821-828 (34 ref.), 1943.
 E – SEC – review – stat. surv. – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans
 – G.I. tract – liver, kidney – respir. – anti-infectants – *CAAAL-0 A-1034.
- The author reviews the literature concerning outbreaks of carbon tetrachloride poisoning in the 10 yr prior to 1943. 58 instances of poisoning are tabulated. 5 of the cases concerned alcoholics, heavy drinkers, or persons who had ingested alcohol prior to or at the time of exposure.
1086. Quevauviller, A., and Bourrinet, P.
 INFLUENCE DE L'ALCOOLISME AIGU ET CHRONIQUE SUR L'ACTIVITÉ DES
 ANESTHÉSIFIQUES LOCAUX CHEZ LE LAPIN ET LE COBAYE. [Influence of acute and

chronic alcoholism on the activity of local anesthetics in the rabbit and the guinea pig].

Thérapie (Paris), 17: 1219-1223 (9 ref.),

1962.

F – ES – SpS – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – nerv. syst. – skel., muscle, skin – anesthetics – *CAAAL-10376-D2 A-1035.

Rabbits and guinea pigs received 1 ml 40% alcohol sol/100 g body wt po. A 0.5% cocaine sol was tested on the corneas of 13 rabbits 1 hr after alcohol ingestion. It was determined that the cocaine application was equal to a 1.8% cocaine sol without alcohol. A 0.2% procaine sol was injected sc into guinea pigs, and it was found that the dose was equal to the effect of a 0.4% procaine sol without alcohol. Rabbits received 0.4 ml of a 40% alcohol sol/100 g/day po for 7-11 weeks, and guinea pigs were given 0.5 ml/100 g/day po for the same period. The results showed that, in rabbits, a 0.5% cocaine sol will produce the effect of a 1% sol after prolonged alcohol consumption. In the guinea pigs, a 0.5% procaine sol was found to be equivalent to a 0.2% sol after prolonged administration of alcohol.

1087. Quevauviller, A., and Bourrinet, P.

ÉTUDE CHEZ L'ANIMAL DE L'INFLUENCE DE L'ALCOOLISATION AIGUE ET CHRONIQUE SUR L'ACTIVITÉ DE QUELQUES ANESTHÉSIIQUES GÉNÉRAUX ET NEURO-SÉDATIFS. [Study in animals of the effect of acute and chronic alcoholization on the action of some general anesthetics and neuro-sedatives].

Anesth. Analg. (Paris), 21: 431-439 (11 ref.),

1964.

F – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – CNS – anesthetics – barbiturates – neoplast. agents – sed., hypnot. – tranquilizers – *CAAAL-11086-D2 A-1036.

Mice received 1 ml 40% ethanol/100 g body wt; exposure within 1 hr to ether produced an average sleep duration of 247 sec, compared to 20 sec for non-ethanol controls. Guinea pigs were given 1 g/kg urethane 1 hr after 1 ml 40% ethanol/100 g, or 24 hr after the last dose of 0.5 ml 40% ethanol/100 g, given 5 times/week for 3 months; the mean duration of sleep was 342 min in the acutely-intoxicated animals, and 404 min in the chronically-intoxicated animals, as compared to 146 min in controls. 3 of 6 rabbits treated with 0.4 ml/100 g ethanol and then given 0.75 g/kg urethane ip died, whereas controls slept for 135 min; the other 3 ethanol-treated animals slept for more than 4 hr. 30 mg/kg nembutal in mice and 50 mg/kg thiopental in mice and rats showed a similar enhancement of toxicity by ethanol. Ethanol pretreatment intensified performance impairment in mice caused by 0.75 mg/kg chlorpromazine, 2 mg/kg reserpine, 0.50 mg/kg butyrophenone, 30 mg/kg meprobamate, and 25 mg/kg methylpentynol carbamate.

1088. Rachek

VNUTRIVENNYI ALKOGOL'NO-GEKSENALOVYI NARKOZ. [Intravenous alcohol-hexobarbital narcosis].

Khirurgiia (Moscow), 11: 30-32 (0 ref.),

1944.

R – general – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – cardiovasc. – CNS – barbiturates – elect., water-bal. agents – *CAAAL-4437-V35 A-1037.

Alcohol-hexobarbital narcosis was used in surgical operations on 15 very weak patients—other methods of anesthesia were considered less practical. 500 g of a 25% alcohol sol was administered iv with 0.5 g hexobarbital at the rate of 50 g/min. Sleep began 1-4 min after administration and lasted for several hr. The appearance of the patients was similar to that of a normal sleeping person. Arousal was even and peaceful. The disadvantage of the method is that there is a rapid onset of local aseptic venous phlebitis, with subsequent venous emptying—this was not an obstacle with the types of operations undertaken. It is concluded that the alcohol-hexobarbital mixture not only has narcotic properties, but also has an indirect influence on the organism as a nutritive and antiseptic substance,

and as one which stimulates cardiac activity. Complications such as excitation, vomiting, and general sluggishness after arousal are absent. Sleep is deep and long-lasting, and anesthesia complete.

1089. Rakieten, N.

THE EFFECTS OF ACETOPHENETIDIN, ACETANILID, AMIDOPYRINE, ANILINE AND PARA-AMINOPHENOL ON THE RATE OF DISAPPEARANCE OF ETHYL ALCOHOL FROM THE BLOOD.

Quart. J. Stud. Alcohol (New Haven), 3(1): 97-102 (15 ref.), 1942.
E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – mammals – acute admin. – in vivo – dose resp. – blood lev. – liver, kidney – metab. proc. – analg., antipyret. – miscellaneous – tranquilizers – *CAAAL-337-A1 A-1038.

64 rats received 1 g alcohol/kg ip and various antipyretics. 11 controls received alcohol only. Acetophenetidin, 200 mg/kg, had no significant effect on the blood alcohol level; 400, 600, and 800 mg/kg reduced the rate of blood alcohol decline. Aniline in a dose of 200 mg/kg had no effect, but 400 mg cut the rate of alcohol disappearance in half. Para-aminophenol, 600 mg/kg, and amidopyrine, 200, 400, and 600 mg/kg, had no effect on the rate of disappearance of alcohol from the blood. 2 human subjects received, on separate occasions, 1.3 g aspirin, 1.3 g acetophenetidin, or 1 g pyramidon, with 120 cc of 45% whiskey. No effect on the rate of disappearance was found.

1090. Ramsey, H., and Haag, H. B.

THE SYNERGISM BETWEEN THE BARBITURATES AND ETHYL ALCOHOL.

J. Pharmacol. Exp. Ther. (Baltimore), 88: 313-322 (14 ref.), 1946.
E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – dose resp. – blood lev. – absorp., distrib., stor. – CNS – barbiturates – sed., hypnot. – *CAAAL-4558-D2 A-1039.

4 sets of experiments were conducted. In 1 experiment, mice received po sodium seconal (100 mg/kg), sodium pentobarbital (130 mg/kg), or sodium barbital (650 mg/kg), either as an aqueous sol or an alcohol sol (4.2 cc 95% alcohol/kg). In another experiment with dogs, 5.3-10.9 mg/kg sodium pentothal was given iv with or without 1.5 cc or 3.0 cc/kg alcohol po. In a third experiment, dogs received 3 cc/kg 95% alcohol po, 180 mg/kg sodium barbital iv, or alcohol plus sodium barbital 1 hr later. In a fourth experiment, rabbits received iv: 1.5 cc/kg alcohol, alcohol plus 30 mg/kg sodium pentobarbital, sodium pentobarbital plus 2 mg/kg picrotoxin, or alcohol plus sodium pentobarbital plus picrotoxin. It was found that the LD₅₀ of sodium seconal in mice was lowered by alcohol. The mortality percentage in mice from standard po doses of sodium seconal, sodium pentobarbital, and sodium barbital was materially increased by alcohol. Alcohol pretreatment reduced the anesthetic dose of sodium pentothal in dogs, and greatly increased the duration of anesthesia. The minimal anesthetic dose of sodium pentothal was unchanged after alcohol, but the duration of anesthesia was increased. The onset of sodium barbital anesthesia was delayed by alcohol, and the duration of anesthesia was increased. Picrotoxin was less efficient as an analeptic in combination with alcohol-sodium pentobarbital, than in combination with sodium pentobarbital alone.

1091. Ransom, F.

ACQUIRED TOLERANCE FOR ALCOHOL IN THE FROG'S HEART.

J. Physiol. (London), 53: 141-146 (3 ref.), 1919.
E – SEC – exp. comp. – other org. – in vitro – cardiovasc. – anesthetics – sed., hypnot. – *CAAAL-2392-A2 A-1040.

In experiments with the isolated frog heart, 1% alcohol sol had little effect, 2% produced depression followed by almost complete restoration, and 3-4% stopped the heart (but gradual restoration followed still under the action of the alcoholic sol). It is noted that no restoration occurs when sol

of chloroform, ether, or chloral (toxicity corresponding to 2% or 3% alcohol) are perfused, a fact which supports the idea that the alcohol tolerance may be an increased power of utilizing alcohol as an energy producer.

1092. Raspopova, T. V., and Zaikonnikova, I. V.

VLIIVANIE SPIRTA NA DYKHANIE NA FONE DEISTVIA MORFINA, URETANA I BARBITURATOV. [Effect of alcohol on respiration under the action of morphine, urethane and barbiturates].

Farmakol. Toksik. (Moscow), 18(6): 27-29 (10 ref.),

1955.

R – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – respir. – analg., antipyret. – barbiturates – neoplast. agents – sed., hypnot. – *CAAAL-7808-D2 A-1041.

Cats and rabbits received iv injections of alcohol in doses of 1.5 or 5 ml/kg in a 20% sol, the injections being made rapidly (2 ml/sec) or slowly (2 ml/20 sec). In rabbits pretreated with 30 mg/kg morphine sc, alcohol almost always had a stimulating effect on respiration. Urethane (1 g/kg) given ip to rabbits and cats resulted in a depression by alcohol of ventilation. Barbiturates given to rabbits (hexenal ip and pentothal iv) resulted in no difference in effect on respiration, when administered alone or with alcohol.

1093. Ratcliffe, F.

THE EFFECT OF CHRONIC ETHANOL ADMINISTRATION ON THE RESPONSES TO AMYLOBARBITONE SODIUM IN THE RAT.

Life Sci. (Oxford), 8(1): 1051-1061 (14 ref.),

1969.

E – exp. cont. – cross-tol. – mammals – acute admin. – chronic admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – CNS – liver, kidney – metab. proc. – barbiturates – *CAAAL-0 B-0549.

Rats received aqueous solutions of ethanol (E), with weekly increments in E concentration, over a period of 7 weeks, and the responses to amylobarbitone sodium (50 mg/kg ip), in terms of induction time and sleeping time, were assessed at the end of each week of E administration and on the third and seventh day after withdrawal. A substantial cross-tolerance to amylobarbitone developed by the end of the first week, with a further decrease in response occurring after the sixth and seventh weeks. An elevated brain amylobarbitone concentration was detected by the sixth week, and returned to control levels 3 days after E withdrawal. An increased iv metabolism of amylobarbitone, as determined by its blood concentration 70 min after injection, was demonstrable throughout the period of E administration and for 7 days after E withdrawal. It is concluded that chronic administration of E decreases appreciably the susceptibility of rats to amylobarbitone, first by a stimulation of drug-metabolizing enzymes (liver), and later by a decrease in tissue sensitivity (brain) to the drug.

1094. Rauch, H. W. M.

ZUR BEHANDLUNG DER AKUTEN ALKOHOLVERGIFTUNG. [The treatment of acute alcohol poisoning].

Med. Klin. (Munich), 45(42): 1348-1350 (0 ref.),

1950.

G – general – case hist. – DC (antidotal) – humans – CNS – G.I. tract – respir. – stimulants – *CAAAL-5661-N14 A-1042.

Analeptic therapy is advocated in the treatment of acute alcohol poisoning. A combination of coramine and cardiazol is reported to have been given in 14 cases of severe alcohol poisoning. The basic dose used was 5 cc of coramine in 25% sol and 4 cc of cardiazol in 10% sol, injected quickly one after the other iv. In all cases, therapy produced sneezing, and, in most cases, vomiting within a few min. The treatment was successful in all cases, with no complications. The question of pathologi-

cal intoxication is discussed. It was considered that to counteract such a condition, stimulants would be beneficial because of the narcotic effect of alcohol. The same medication was therefore tried in 2 cases of pathological intoxication without success. This shows that, in pathological intoxication, alcohol is perhaps only a means of releasing abnormal reactions in the brain; it is not an intoxication in the usual sense of the word.

1095. Rauschke, J.

ÜBER DIE EIGNUNG VON „ALKOHOL MINUS“ (ALMI) ALS

ERNÜCHTERUNGSMITTEL. [The effectiveness of “Alcohol minus” (ALMI) as a sobering-up agent].

Blutalkohol (Hamburg), 5(4): 221-228 (12 ref.),

1968.

G – exp. comp. – DC (decrease) – humans – acute admin. – in vivo – metab. proc. – unclass. ther. agents – *CAAAL-0 B-0550.

To determine the effect of “alcohol minus” (ALMI), a purported sobering-up compound, a series of experiments were conducted. 12 men and 2 women were divided into 3 groups. The first group consumed alcohol with ALMI, the second drank alcohol with a prepared liquid of similar structure, and the third drank alcohol plus a commercial fruit drink. The structure of ALMI was found to be 20% honey, with 80 g/l fructose, 80 g/l glucose, 1.8 g/l acetic acid, traces of carbonic acid, and an unknown acid, making a total acid content of 4.9 g/l. The liquid of similar structure was prepared from 1 lb of honey, 2 liters of water and 2-1/2 lemons. After each session, the subjects received a blood test, and the results were tabulated. The results showed that the effect of ALMI (consumed with an unstated amount of alcohol) was the same as that of the other liquids. Nor was it found to be true that ALMI increases the decomposition of alcohol, since it merely decreased the time of absorption. ALMI will undoubtedly disappoint expectations of the public to use it as a sobering-up drug or to prevent alcoholic intoxication, as it is concluded that any kind of food or non-alcoholic beverage would have the same effect.

1096. Ravina, A., and Voisin, J.

CONCEPTIONS RÉCENTES SUR LA PATHOGÉNIE ET LE TRAITEMENT DE

L'INTOXICATION PAR L'ALCOOL MÉTHYLIQUE. [Recent theories on the pathogenesis and treatment of methyl alcohol poisoning].

Presse Med. (Paris), 53(28): 384-385 (5 ref.),

1945.

F – review – DC (antidotal) – humans – mammals – other drug lev. – acid-base, blood pH, elect. – metab. proc. – senses – alcohols – *CAAAL-4461-E4 A-1043.

This article is a review of literature on methanol poisoning, and an extensive discussion of Røe's study (Acta Med. Scand., 113: 558-608, 1943). Røe, having noted the absence of severe symptoms of methyl alcohol poisoning in persons who had immediately ingested strong doses of ethyl alcohol, undertook some clinical studies which tended to prove that ethyl alcohol is an antidote to methyl alcohol poisoning. This conclusion appears to have been reached previously in the investigations on dogs by Asser (Zeitschrift für Experimentelle Pathologie und Therapie, 15: 322-334, 1914).

1097. Raynes, A. E., and Ryback, R. S.

EFFECTS OF ALCOHOL AND CONGENERS ON AGGRESSIVE RESPONSE IN “*BETTA SPLENDENS*”.

Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 130-135 (16 ref.),

1970.

E – exp. cont. – exp. comp. – congen. stud. – other org. – acute admin. – in vivo – mot. perform. – psychol. perform. – species or sex diff. – CNS – metab. proc. – *CAAAL-12894 B-0551.

The aggressive response of male Siamese fighting fish, *Betta splendens*, as measured by the accumulated time of gill membrane lowering in response to a 3 min exposure to their mirror images,

was compared before and 6 hr after immersion in 1 of the following sol: 285 mg ethanol/100 ml, bourbon diluted to the same ethanol concentration, or a congener sol diluted to contain the same amount of congeners as the bourbon sol, and containing 10 mg ethanol/100 ml. The mean aggressive responses for each group of 12 fish after 6 hr were significant: a 11.42 sec increase in ethanol sol, and a decrease of 24.75 and 19.33 sec, respectively, in bourbon and congener sol. 5 of 12 fish in congener sol showed no aggressive response, whereas all the fish in bourbon sol showed some residual aggression, indicating that the congener sol had a more profound depressive effect. The tendency for congeners to depress the aggressive reaction more than bourbon is perhaps due to the stimulating effect of the ethanol content of bourbon being masked by the overall depressant effect of the congeners. It is believed that congeners and ethanol involve different physiological mechanisms, since bourbon showed no summation effect.

1098. Reddy, D. G., Reddy, D. B., and Prabhaker, V.

AN EXPERIMENTAL STUDY OF THE RESPIRATORY TRACT OF MICE EXPOSED TO (1) TOBACCO SMOKE AND (2) TOBACCO SMOKE AND ALCOHOL VAPOUR.

Indian J. Path. Bact. (Bombay), 11(1): 13-18, + 2 pp of pictures (10 ref.), 1968.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. – in vivo – respir.
– *CAAAL-0 B-0424.

A study was undertaken to see if vapours of alcohol, gasoline, and diesel oils could function as promoting agents and produce neoplastic changes in the respiratory tract of mice simultaneously exposed to tobacco smoke. Fifty mice were divided into 4 groups: group 1 was exposed to cigar smoke, group 2 to alcohol vapours, group 3 to alcohol and cigar smoke, and group 4 was kept as control. It was found that exposure to tobacco smoke alone daily for 12 months failed to show any significant pathological changes, but, when alcohol vapour was administered simultaneously, the bronchial epithelium on occasions showed benign dysplasia and squamous metaplasia filling the entire lumen of the bronchioles. Experimental details and the role of alcohol in the production of these changes are discussed in detail.

1099. Redetzki, H. M.

THE INTERACTIONS OF MONOAMINE OXIDASE INHIBITORS (MAOI) WITH ALCOHOL: BIOCHEMICAL AND PHARMACOLOGICAL ANALYSIS.

Fed. Proc. (Bethesda), 26(2): 616 (0 ref.), 1967.
E – abst. – exp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin.
– in vivo – in vitro – blood lev. – mot. perform. – species or sex diff. – CNS – metab. proc. – enzymes
– *CAAAL-0 B-0425.

It was shown that, while monamine oxidase inhibitors (MAOI) of the hydrazine and monohydrazine types inhibited the activity of liver alcohol dehydrogenase in vitro, the rates of alcohol oxidation in vivo varied with the examined species. Tranylcypromine, 5-20 mg/kg, significantly decreased the rate of decline of blood alcohol and total body alcohol in mice, but did not affect the alcohol metabolism in rabbits. Blood acetaldehyde levels were slightly increased, but not to an extent comparable with a disulfiram reaction. It is concluded that incompatibility reactions are probably related to a potentiation of catecholamine actions, rather than to an interference with alcohol metabolism.

1100. Redetzki, H. M.

DRUG-ALCOHOL INTERACTIONS.

J. Louisiana Med. Soc. (New Orleans), 120(12): 471-476 (12 ref.), 1968.
E – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – mammals
– blood lev. – mot. perform. – psychol. perform. – species or sex diff. – cardiovasc. – CNS – G.I.
tract – metab. proc. – anti-infectants – autonomic agents – barbiturates – cardiovasc. agents – elect.,
water-bal. agents – enzymes – hormones, hormone antag. – musculoskel. agents – sed., hypnot. –
tranquilizers – unclass. ther. agents – *CAAAL-0 B-0426.

In this general discussion of the problem of drug-alcohol interactions, the author covers the following combinations with alcohol: drugs used in aversion treatment of alcoholism, drugs which cause incompatibility reactions (including monamine oxidase inhibitors), sedatives and tranquilizers, and agents used to accelerate alcohol metabolism. It is pointed out that 2/3 of the population of the United States above 15 yr of age drink alcohol, and that it had been estimated that, at any point in time, 10-20% of drivers are taking medication resulting from a prescription.

1101. Redmond, G., and Cohen, G.

INDUCTION OF LIVER ACETALDEHYDE DEHYDROGENASE: POSSIBLE ROLE IN ETHANOL TOLERANCE AFTER EXPOSURE TO BARBITURATES.

Fed. Proc. (Bethesda), 29(2): 275 Abs (0 ref.),

1970.

E – abst. – exp. cont. – exp. comp. – mammals – chronic admin. – in vivo – metab. proc. – barbiturates – *CAAAL-0 B-0974.

Paris RIII and C57BL mice received saline, phenobarbital (75 mg/kg), or ethanol (2.4 g/kg) ip 2 times/day for 4 days. The activity of acetaldehyde dehydrogenase (AcDH) in liver homogenates was measured spectrophotometrically by following the appearance of NADH in the presence of acetaldehyde. In some experiments, pyrazole (0.001 M) was added to inhibit alcohol dehydrogenase. It was found that phenobarbital induced AcDH by 100% ($p < 0.01$), whereas ethanol did not induce AcDH. It is suggested that, in part, the more rapid removal of the toxic metabolite, acetaldehyde, may be the basis for the tolerance to alcohol exhibited by regular users of barbiturates.

1102. Redmond, G., and Cohen, G.

INDUCTION OF LIVER ACETALDEHYDE DEHYDROGENASE: POSSIBLE ROLE IN ETHANOL TOLERANCE AFTER EXPOSURE TO BARBITURATES.

Science (Washington), 171(3969): 387-389 (15 ref.),

1971.

E – exp. cont. – cross-tol. – mammals – acute admin. – in vivo – absorp., distrib., stor. – liver, kidney – metab. proc. – barbiturates – *CAAAL-0 B-0975.

Mice were injected ip 2 times/day for 4 days with saline, phenobarbital (75 mg/kg), or ethanol (2.4 g/kg), and were then killed on day 5. Acetaldehyde dehydrogenase (AcDH) in liver homogenates was measured. After phenobarbital, the AcDH level was double that in controls, but was not affected by ethanol. There was no significant difference in alcohol dehydrogenase activity between control and phenobarbital-treated mice, showing that the apparent increase in AcDH was not due to a decrease in alcohol dehydrogenase activity. Other tests showed that AcDH activity could not be attributed to a decreased rate of removal of NADH. Since the formation of acetaldehyde from ethanol is generally considered to be the slowest, rate-limiting step in ethanol metabolism, the increased AcDH activity induced by phenobarbital cannot be expected to lower substantially the over-all rate of ethanol disappearance; however, it should result in lower acetaldehyde levels in tissues. This could be a contributory factor to the cross-tolerance of barbiturate-users to ethanol. The finding of Rubin and Lieber (Science, 159(3821): 1460-1470, 1968) that, in rats and humans, ethanol induces the liver enzymes that metabolize barbiturates—suggesting that this contributes to the cross-tolerance of alcoholics for barbiturates—indicates that enzyme induction may underlie both sides of cross-tolerance.

1103. Regan, T. J., Koroxenidis, G., Moschos, C. B., Oldewurtel, H. A., Lehan, P. H., and Hellems, H. K.

THE ACUTE METABOLIC AND HEMODYNAMIC RESPONSES OF THE LEFT VENTRICLE TO ETHANOL.

J. Clin. Invest. (Cincinnati), 45: 270-280 (64 ref.),

1966.

E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – cardiovasc. – metab. proc. – skel., muscle, skin – barbiturates – *CAAAL-12326-D2 B-0976.

0.1 ml/kg/min of 15% ethanol were infused iv for 2 hr in intact dogs anesthetized with 3 mg/kg morphine and 12 mg/kg pentobarbital. A decreased myocardial efficiency after alcohol administration in dogs under pentobarbital anesthesia, noted by other researchers, was observed. Diminished left ventricular function, manifested as a decline of stroke output and rise of end-diastolic pressure, was observed as early as 15 min, was well-established by 30 min at a blood alcohol concentration of 110 ± 13 mg/100 ml, and persisted for 5 hr without significant heart rate or arterial pressure changes. Evidence of cardiac tissue injury was observed after 90 min in the significant elevations of coronary sinus potassium and phosphate ion concentrations, and in the transaminase enzyme increase. Triglyceride content of the myocardium increased from 0.38 to 1.25 mg/g wet wt. The authors point out that sympathetic stimulation may be involved, since infusion of catecholamines provoked similar effects.

1104. Reid, C. H., Jr.

THE SYNERGISTIC EFFECT OF ACETONE AND ALCOHOL IN THE PRODUCTION OF SYMPTOMS OF COMA.

North Carolina Medical Journal (Winston-Salem), 6: 416-417 (3 ref.), 1945.
E - exp. cont. - DC (unchanged) - mammals - acute admin. - in vivo - CNS - hormones, hormone antag. - miscellaneous - *CAAAL-4282-B2 A-1044.

Mice were divided into 4 groups. Group 1 (7 mice) received acetone ip in saline; group 2 (5 mice) received ip 1.0 to 2.6 cc 9.5% alcohol; group 3 (2 mice), a combination of acetone (0.9 and 1.0 cc 10% acetone) and 1.2 cc 9.5% alcohol; and group 4 (3 mice) received saline alone. Results ranging from death to prompt recovery are given in a table. It is concluded that alcohol does not increase the potency of acetone, but simply acts as an additional ketone body. The tolerance of the diabetic patient for alcohol will be reduced by the extent of any pre-existing acidosis. A mixture of 1/2 the maximal sublethal dose of each substance affected the state of consciousness in approximately the same degree as did the injection of 1 maximal sublethal dose of either substance alone.

1105. Reifenstein, E. C., Jr., and Davidoff, E.

THE TREATMENT OF ALCOHOLIC PSYCHOSES WITH BENZEDRINE SULFATE: PRELIMINARY REPORT.

J.A.M.A. (Chicago), 110(22): 1811-1812 (12 ref.), 1938.
E - exp. - DC (antidotal) - humans - acute admin. - in vivo - CNS - amphetamines - *CAAAL-912-N13 A-1045.

20 patients with pathological intoxication, delirium tremens, acute hallucinosis, and Korsakoff's psychosis were given 10 to 30 mg of benzedrine po or iv. A definite acceleration of improvement was seen in 93% of the patients. In states of alcoholic intoxication in which no psychosis was demonstrable, benzedrine sulfate usually produced an even more satisfactory response than in states of intoxication in which a psychosis existed. The headache, fatigue, languor, and mental retardation which are characteristic of a "hangover" usually disappeared within an hr after a morning dose of from 5 to 10 mg. The authors suggest that benzedrine sulfate may produce these beneficial responses in alcoholic states through its action in stimulating the central and the sympathetic nervous system, and also directly by neutralizing or antagonizing the alcohol itself.

1106. Reifenstein, E. C., Jr., and Davidoff, E.

THE USE OF AMPHETAMINE (BENZEDRINE) SULFATE IN ALCOHOLISM WITH AND WITHOUT PSYCHOSIS.

New York J. Med. (New York), 40: 247-254 (12 ref.), 1940.
E - exp. cont. - DC (antidotal) - humans - psychot. humans - drug-dep. humans - acute admin. - in vivo - psychol. perform. - CNS - amphetamines - *CAAAL-1239-N13 A-1046.

The use of amphetamine sulfate (20-30 mg/day) was evaluated in the treatment of psychotic and non-psychotic alcoholics. The drug was found to be of value in the more acute phases of alcoholism, with and without psychosis, and unsatisfactory in the treatment of alcohol addiction and cases of protracted alcoholic psychoses. Amphetamine sulfate was found to be very effective in treating the acute phases of alcoholic intoxication. Intoxicated, stuporous persons were aroused within 30 min, following an iv injection of 20 to 30 mg amphetamine sulfate; in persons who received this drug in the acute stage of intoxication, the characteristic physiological and psychological after-effects and withdrawal symptoms were aborted or mild.

1107. Reifenstein, E. C., Jr.

AMPHETAMINE SULFATE-ETHYL ALCOHOL ANTAGONISM IN THE RABBIT.

J. Lab. Clin. Med. (St. Louis), 27(2): 131-139 (6 ref.), 1941.
E – exp. cont. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – absorp., distrib., stor. – CNS – amphetamines – *CAAAL-3451-D2 A-1047.

423 rabbits received sol of amphetamine sulphate and/or ethanol by stomach tube. It was found that the median min lethal dose (MLD₅₀) of 25% alcohol was 7.25 g/kg, and the MLD₅₀ of 0.25% amphetamine was 85 mg/kg. The MLD₅₀ of mixture A (1 MLD₅₀ of alcohol plus 1/2 MLD₅₀ amphetamine/unit vol) was 6.025 g/kg alcohol with 35.32 mg/kg amphetamine. The MLD₅₀ of mixture B (1 MLD₅₀ of alcohol plus 1 MLD₅₀ of amphetamine/unit vol) was 5.78 g/kg alcohol with 67.62 mg/kg amphetamine. The MLD₅₀ of mixture C (1/2 MLD₅₀ of alcohol plus 1 MLD₅₀ of amphetamine/unit vol) was 5.329-5.968 g/kg alcohol with 125-140 mg/kg amphetamine. The addition of 5 g/kg alcohol alone produced narcosis with an onset of 17.4 min and a duration of 408 min, whereas, in combination with 1 MLD₅₀ (85 mg) amphetamine/kg, the onset was 55.9 min and the duration was 307 min. It is concluded that, in the rabbit, amphetamine has an analeptic effect on the narcosis of moderate amounts of alcohol, but has no analeptic action on the narcosis of lethal doses, and even increases the toxicity of near-lethal quantities. Alcohol protects the animal against 1.5 to 2 times the MLD₅₀ of amphetamine.

1108. Reinartz, E. F. K.

ÜBER DIE EINWIRKUNG VON MEDIKAMENTEN BEI 500

KRAFTFAHRZEUGUNFÄLLEN IN FRANKFURT A.M. [The influence of drugs on 500 car accidents in Frankfurt a.M.].

Dissertation, Medical Faculty of the University of Mainz, West Germany, 32 pp. (91 ref.),

1962.

G – stat. surv. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – mot. perform. – CNS – analg., antipyret. – barbiturates – *CAAAL-0 A-0057.

The author considers the use and abuse of alcohol and drugs in relation to automotive medicine. Statistical data on traffic accidents in Frankfurt, and percentages of drug and alcohol users are given. The effect of interactions between alcohol and drugs are discussed and illustrated. In 1 of the cases cited, 2 glasses of brandy, one luminal tablet (0.15 g), and an analgesic (dolviran) resulted in synergism which induced sleep while driving a motor vehicle. In another case, brandy, beer, and 3 sobering-up tablets ("cafaspin") caused appreciable intoxication, resulting in a traffic accident. Public education of interaction dangers between alcohol and drugs, especially analgesics, is needed.

1109. Reinert, R. E.

A COMPARISON OF RESERPINE AND DISULFIRAM IN THE TREATMENT OF ALCOHOLISM.

Quart. J. Stud. Alcohol (New Haven), 19(4): 617-622 (1 ref.), 1958.

E – exp. comp. – DC (unspec.) – drug-dep. humans – acute admin. – in vivo – psychol. perform. – CNS – tranquilizers – unclass. ther. agents – *CAAAL-8053-M3 A-1048.

A comparative study was carried out on reserpine (3-4 mg/day) and disulfiram (0.5 g/day), with respect to their relative effectiveness in treating the chronic alcoholic. Hospital stay was 2-3 months. Follow-up was attempted for 13 months, and treatment evaluated from the patient's record of sobriety and his testimony as to the effect of the drug on psychic distress. Disulfiram was found to be more effective. The reaction of a patient who had reverted to drinking while on reserpine is noted. He complained of increased tension and insomnia.

1110. Reinhard, J. F., and Spector, E.

EFFECT OF PHENOBARBITAL, PHENYLBUTAZONE, 3,4-BENZPYRENE, OR 3-METHYLCHOLANTHRENE ON ETHANOL METABOLISM IN THE RAT.

Toxic. Appl. Pharmacol. (New York), 17(1): 12-22 (7 ref.),

1970.

E – exp. cont. – DC (decrease) – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – in vitro – blood lev. – CNS – liver, kidney – metab. proc. – analg., antipyret. – barbiturates – *CAAAL-0 B-0977.

The effects of phenobarbital (Ph), phenylbutazone (Pb), 3,4-benzpyrene (B), and 3-methylcholanthrene (M), on the hypnotic and toxic effects of ethanol in intact rats, and on ethanol metabolism in rat liver homogenates, were investigated. Ph, Pb, M, and B were administered ip to groups of 15-20 immature male rats (35-40 g) on 3 successive days, in doses of 38, 76, and 150 mg/kg/day; 125 mg/kg/day; 125 and 250 mg/kg/day; and 25 mg/kg/day, respectively. 24-48 hr after the last pretreatment injection, 5 test and 5 control animals were sacrificed and ethanol metabolism tested in liver homogenate preparations. The remaining animals were challenged with 1.0 ml/100 g 40% (v/v) ethanol ip, and sleeping times were recorded. The results showed a significant rise in in vitro ethanol metabolism by liver homogenates after Ph and M pretreatment, while B and Pb were ineffective. Ethanol-induced sleeping time was significantly decreased by Ph, B, and M, but not by Pb. The acute toxicity of ethanol was markedly reduced by drug pretreatment from the control value of 60-80% mortality to 17-29% test group mortality; however, plasma concentrations of ethanol were identical in both groups. It is concluded that some mechanism other than increased ethanol metabolism was responsible for the decreased sleeping time and toxicity. This may involve reduced brain cell sensitivity or altered permeability of cell membranes to ethanol.

1111. Reisby, N.

KOMBINATIONSVIRKNINGEN AF ALKOHOL OG PSYKOFARMAKA: EN GENNEMGANG AF 15 ÅRS SAGER FORELAGT RETSLAEGERÅDET. [The combined effect of alcohol and psychopharmacological drugs: a review of cases submitted to the Medico-Legal Council in a 15 year period].

Ugeskr. Laeg. (Copenhagen), 129(4): 109-117 (6 ref.),

1967.

Da – ES – stat. surv. – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev. – antidepressants – barbiturates – sed., hypnot. – tranquilizers – *CAAAL-0 B-0427.

Case material of 182 individuals, 20 to 65 yr of age, collected from 1950 to 1964 by the Medico-Legal Council in Denmark, is reviewed. Graphs are plotted, indicating an appreciable increase in combining drugs with alcohol. In all, 258 combinations are tabulated in which the dose effect of a particular drug on blood alcohol levels was indicated. Meprobamate-alcohol combinations figure prominently. The mean blood alcohol level was lower in 37 cases involving all degrees of intoxications in which combinations of drugs and alcohol were assumed, as compared to 67 cases where alcohol was assumed to be the sole cause of intoxication. In this study, 93% of the cases involved traffic violations, and 13 cases involved criminal offences. 8 mortalities (100% of the fatal cases) were attributed to synergism between alcohol and drugs.

1112. Reisby, N., and Theilgaard, A.

INDLEDENDE UNDERSØGELSER OVER KOMBINATIONSVIRKNINGEN AF ALKOHOL OG MEPROBAMAT VED HJÆLP AF NOGLE

OPMAERKSOMHEDSPRØVER. [Preliminary investigations of the combined effect of alcohol and meprobamate employing concentration tests].
 Ugeskr. Laeg. (Copenhagen), 129(4): 118-122 (3 ref.), 1967.
 Da – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – psychol. perform. – CNS – tranquilizers – *CAAAL-0 B-0428.

6 human subjects received, at 8-day intervals, 800 mg meprobamate, enough alcohol to produce a blood alcohol level of 1°/oo, and meprobamate plus alcohol, under a double-blind procedure. 3 concentration tests and a time assessment were applied. Deviation from the mean value of 2 preliminary tests was employed as a rough estimate for the relative extent of the influence of the administrations. In 66 out of 72 tests, the deviation was greatest with alcohol-meprobamate. There were wide variations in the individual effects, variations which were probably due to personality variables. The results do not allow a definite statement about the nature of the combined effect; however, they support the presumption of the synergistic effect, and illustrate the risk involved in simultaneous ingestion of alcohol and tranquilizers of the meprobamate type.

1113. Reisby, N., and Theilgaard, A.

THE INTERACTION OF ALCOHOL AND MEPROBAMATE IN MAN.

Acta Psychiat. Scand. (Copenhagen), Suppl. 208: 207 pp. (190 ref.), 1969.
 E – exp. cont. – review – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – species or sex diff. – cardiovasc. – CNS – senses – skel., muscle, skin – tranquilizers – *CAAAL-0 B-0548.

The combined effect of alcohol and meprobamate on psychomotor behavior was studied in 64 student volunteers. On 4 occasions at weekly intervals, 53 subjects were administered: 1) 2 placebo tablets, 2) 1.1 g alcohol/kg body wt, 3) 800 mg meprobamate, or 4) alcohol plus meprobamate in the above doses. 11 subjects served as independent controls. Before and approximately 1 1/2 and 4 1/2 hr after drug ingestion, the subjects completed a battery of psychological and psychomotor tests. Following the administration of alcohol and meprobamate together, a statistically significant combined effect was evident in increased overestimation of time, decreased fixation stability of the eyes, increased reaction times, and increased error in the distributor test (attention). A combined effect was also suggested in Maddox Wing (ocular coordination), 1-hand tracing, standing steadiness, blood pressure, and pulse rate determinations. Although the subjects reported no difference between the subjective effects of alcohol and alcohol plus meprobamate, the authors observed a reduction in the degree of alertness, and an increase in the number of unusual, eccentric reactions after combined intake. It is concluded that the combined effect of alcohol and meprobamate is determined to a marked degree by the personalities of the subjects and the situational contexts, and it appears to be additive.

1114. Reisby, N.

PERSONALITY STRUCTURE AND THE INTERACTION OF ALCOHOL AND MEPROBAMATE.

In: *Collegium Internationale Neuro-Psychopharmacologium (C.I.N.P.), Abstracts. II*. Seventh International Conference, Prague, Czechoslovakia, August 11-15, 1970. Prague: C.I.N.P., p. 360 (0 ref.), 1970.
 E – abst. – exp. cont. – exp. comp. – DC (unspec.) – humans – acute admin. – chronic admin. – mot. perform. – psychol. perform. – CNS – tranquilizers – *CAAAL-0 B-0580.

60 subjects (11 women) were given various attention and motor-coordination tests under 4 conditions (double-blind administration): 1) placebo, 2) 1.1 g alcohol/kg, 3) 1600 mg meprobamate, or 4) (2) plus (3), according to a latin square technique. 20 of the subjects were pretreated by daily administration of 1600 mg meprobamate for 2 weeks prior to the experiment. The subjects were selected by the Taylor Manifest Anxiety Scale (TMAS), and judged by clinical criteria. TMAS groups with scores of less than 5 and more than 22, and 4 clinical groups with increasing psychopathology were

compared. Using statistical analysis of variance, no differences between groups could be distinguished before or after drug administration, but consistent pre-drug differences were found with non-parametric tests. After dose administration, the greatest change from the pre-drug level was found in the most normal clinical group and in the low scorers on the TMAS.

1115. Rejsek, K.

M-DINITROBENZENE POISONING: MOBILISATION BY ALCOHOL AND SUNLIGHT.

Acta Med. Scand. (Stockholm), 127(1-2): 179-191 (23 ref.), 1947.

E – general – DC (add., infra-add., unspec. incr.) – humans – blood comp., sites, lymph – cardiovasc.

– G.I. tract – respir. – miscellaneous – *CAAAL-4834 A-1049.

Several cases of subchronical m-dinitrobenzene poisoning are described. The poisonings became acute under the influence of alcohol or sunlight. The effect of alcohol had been previously established, and employees working with aromatic nitro- and amino-compounds had always been forbidden, under penalty of dismissal or transfer, to drink alcohol. The author diagnosed the cases by alcohol mobilization (resulting in the appearance of cyanosis and of subjective symptoms) with 2% beer. The cases were remarkable for the length of the latent period—in 1 case, ingestion of a small quantity of beer 6 weeks after the disappearance of all symptoms of acute poisoning produced a serious relapse which could be reproduced in full, 10 days later.

1116. Remmer, H.

DRUG TOLERANCE.

In: Mongar, J. L., et al., eds. *CIBA Foundation Symposium on Enzymes and Drug Action*. London: J. and A. Churchill Ltd., pp. 276-298 (33 ref.), 1962.

E – SEC – exp. comp. – presentation – DC (unchanged) – mammals – acute admin. – in vivo – in

vitro – CNS – metab. proc. – analg., antipyret. – barbiturates – *CAAAL-0 A-1050.

The activating effect of pretreatment with various drugs, including ethanol, on the drug-metabolizing enzymes in rats was studied. The in vitro tests used for enzyme activity were the oxidation of hexobarbitone and the demethylation of methylaminoantipyrine by liver supernatant; the in vivo test for the approximate speed of the oxidation was the sleeping time after administration of hexobarbitone and eunarcon. Ethanol, 6000 mg/kg administered sc, failed to affect either test, and hence failed to accelerate oxidative drug metabolism.

1117. Remmer, H.

INFLUENCIA DEL ETANOL SOBRE EL METABOLISMO DE LAS DROGAS. [Effect of ethanol on drug metabolism].

Archivos de Biología y Medicina Experimentales (Santiago de Chile), 6: 85-88 (4 ref.), 1969.

Sp – exp. cont. – cross-tol. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic

admin. – in vivo – in vitro – blood lev. – CNS – metab. proc. – analg., antipyret. – barbiturates –

*CAAAL-0 B-0429.

Examples are cited, indicating that alcohol may either accelerate or inhibit the oxidation of drugs, and thus reduce or potentiate their action. Rats treated with ethanol (6 g/kg/day for 8-14 days) prior to hexobarbital (100 mg/kg/ip) showed considerable diminution of the duration of anesthesia; this was attributed to cross-tolerance. Ethanol-pretreated rats, given eunarcon or phenobarbital (76 mg/kg/ip) showed a marked difference in sleeping time from the controls. Moreover, ethanol (3.2 mg/kg), given to male rats 12 hr prior to 40 mg/kg phenazone po, inhibited the metabolism of the latter. Excretion of norphenazone was significantly reduced during the first 5-6 hr. The results of in vivo and in vitro tests are plotted and tabulated.

1118. Rentschler, E.
BLUTALKOHOL UND MEDIKAMENTE. [Blood alcohol and drugs].
Angew. Chem. (Weinheim), 71(3): 131 (0 ref.), 1959.
G – abst. – general – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – blood lev. – metab.
proc. – barbiturates – unclass. ther. agents – *CAAAL-0 A-1051.

The sensitizing action of tetraethylthiuram disulfide and the catabolism of alcohol are briefly discussed. Similar effects (headache, fatigue, apathy, and aversion towards alcohol) were seen after the intake of an alcohol-irgapyrin combination, and after other pyrazolone agents. The seriousness of an alcohol-barbiturate combination is stressed, because of the possible synergistic action. No changes in the blood alcohol curve were seen after barbiturate application. Higher doses probably lead to decreased absorption and oxidation of alcohol.

1119. Reynolds, W. A., and Lowe, F. H.
MUSHROOMS AND A TOXIC REACTION TO ALCOHOL: REPORT OF 4 CASES.
New Eng. J. Med. (Boston), 272(12): 630-631 (6 ref.), 1965.
E – general – case hist. – DC (sensit.) – humans – cardiovasc. – *CAAAL-0 B-0552.

4 adults ate a large number of mushrooms at the evening meal, the majority of the fungi being inky caps (*Coprinus atramentarius*). The next day, each person consumed a fresh bottle of beer, and each became immediately ill. Symptoms and signs included: a profound flushing of the face, a metallic taste in the mouth, paresthesia of the extremities, palpitation and tachycardia, a swelling feeling in the hands, nausea and vomiting, and normal reflexes. Further examination in hospital revealed erythema of the neck and extremities, pulse variation from 92 to 110, confusion, and a feeling of excessive warmth. Recovery was short and uneventful. Other persons who ate the mushrooms but drank no beer suffered no ill effects. 1 of the 4 persons drank 2 beers 3 hr after the mushroom meal, and noted only a mild tingling of the extremities. The mushroom-beer effects are the same as those of disulfiram followed by alcohol, but the former may vary in intensity, according to the time interval between mushroom and alcohol ingestion.

1120. Richards, V.
THE INTRAVENOUS INJECTION OF AMMONIA IN ACUTE ALCOHOLISM.
Lancet (London), 1: 115 (0 ref.), 1880.
E – general – case hist. – DC (antidotal) – humans – CNS – stimulants – *CAAAL-0 A-1052.

The author agrees with Robert Hamilton (Lancet (London), 2: 157-158, 1879) that ammonia is useful in the treatment of acute alcoholic poisoning. He recounts a controversy which he provoked by stating that the purported efficacy of ammonia in the treatment of snake bite is due to the fact that many of the symptoms which were supposed to have been caused by snake venom were in fact caused by alcoholic poisoning, owing to the quantity of alcohol given to the patient. To illustrate the point, he mentions the case of a man who had been bitten by a snake and was given 1 1/2 bottles of brandy in 3 hr; he was perfectly comatose, and his pupils were contracted. 10 minims of ammonia were injected iv, and the patient, "woke up at once, and recognized friends and walked about."

1121. Riedler, G.
EINFLUSS DES ALKOHOLS AUF DIE ANTIKOAGULANTIENTHERAPIE. [Influence of alcohol on anticoagulant therapy].
Thromb. Diath. Haemorrh. (Stuttgart), 16: 613-635 (33 ref.), 1966.
G – ES – FS – exp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood comp., sites, lymph – cardiovasc. – liver, kidney – *CAAAL-0 B-0430.

10 patients were examined after wine consumption, and 2 after whiskey. All patients were treated with anticoagulants. It was observed that the factors II, VII, X, and the prothrombin time of "quick" were

later followed by a distinct, and, in certain cases, even impressive decrease of factors VII and X, while the decrease of factor II and the prothrombin time of "quick" was less pronounced. A possible explanation is discussed. It is concluded that alcohol consumption should be avoided during anticoagulant therapy, since factors VII and X are easily affected and decline several times below the critical values. This is particularly important in the case of older patients with a possible liver deficiency and a slight heart insufficiency.

1122. Rietbrock, N., and Säger, U.

EINFLUSS INTERMITTIERENDER ÄTHANOLGABEN AUF DEN METHANOLUMSATZ IM HUNDE. [Influence of intermittent doses of ethanol on the methanol metabolism of the dog].

Naunyn Schmiedeberg. Arch. Pharm. Exp. Path. (Berlin), 257(1): 57 (1 ref.), 1967.
G – exp. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – acid-base, blood pH, elect. – metab. proc. – alcohols – *CAAAL-0 B-0431.

The time of application and duration of ethanol administration in methanol poisoning and the effect on metabolic acidosis were investigated. In experiments with dogs, it was observed that ethanol inhibits the formation of metabolic acidosis. Even existing acidosis disappears after application of ethanol. Treatment is only successful if a constant blood alcohol level of 0.5-0.8°/oo is maintained for 4-5 days. At the beginning of the treatment, combined doses of ethanol and folic acid show the fastest effect.

1123. Rietbrock, N.

ANTIDOTTHERAPIE DER METHYLALKOHOLVERGIFTUNG. [Antidotal therapy of methanol intoxication].

Arch. Toxik. (Berlin), 24(1): 56-70 (23 ref.), 1968.
G – exp. cont. – DC (antidotal) – DC (decrease) – mammals – acute admin. – in vivo – metab. proc. – alcohols – elect., water-bal. agents – nutritive agents – *CAAAL-0 B-0432.

A review of literature is given on the antidotal therapy of poisoning by methanol. 0.16 M sodium bicarbonate iv, 24-48 hr after methanol intoxication, or 0.3 M THAM-buffer (and pretreatment in either case with 0.75 mg/kg amethopterin), administered to dogs, decreased the toxicity of formic acid. The efficacy of ethanol therapy (0.75 g/kg, then 0.22 g/kg/1 1/2 hr) was tested in vivo on dogs under controlled conditions. The results (plotted) show that ethanol retarded the elimination of methanol in plasma compared to the control. Based on the tests and evaluations, the following treatment is recommended for methanol intoxication: ethanol iv to reach a plasma level of at least 0.5°/oo, to be maintained for 2-5 days; folic acid iv in a dose of 10 mg/kg; and, for metabolic acidosis, repeated feeding of THAM buffer or sodium bicarbonate in proportionate doses.

1124. Rietbrock, N.

KINETIK UND WEGE DES METHANOLUMSATZES. [Kinetics and pathways of methanol metabolism].

Naunyn Schmiedenberg. Arch. Pharm. Exp. Path. (Berlin), 263(1): 88-105 (53 ref.), 1969.
G – SEC – general – DC (antidotal) – humans – mammals – absorp., distrib., stor. – liver, kidney – metab. proc. – respir. – alcohols – *CAAAL-0 B-0978.

An extensive discussion is given on the kinetics of methanol oxidation, the elimination of methanol through alcohol dehydrogenase (ADH) and catalase, oxidation through microsomal enzymes, and the elimination of formic acid. The main route of methanol elimination is by oxidation to formaldehyde, which is then converted to formic acid, and finally to carbon dioxide. With respect to ADH and catalase activity, a 90% inhibition of methanol oxidation can be expected theoretically; however, in vivo and in vitro experimental results have not reached this percentage. Since methanol does not effect

ethanol oxidation, and since 1-propanol and 1-butanol inhibit ethanol, but not methanol, oxidation, the inhibition of methanol oxidation by ethanol cannot be considered competitive. On the other hand, ethanol in plasma, even in a concentration of only 0.5-1.0°/oo, completely inhibits the accumulation of formic acid in vivo; this corresponds to a 50-60% inhibition of methanol oxidation by liver microsomes. From this parallel, it is concluded that methanol elimination is carried out predominantly, if not entirely, by liver microsomes.

1125. Rietbrock, N., Herken, W., and Heberlein, W.

UNTERSCHIEDLICHE BEEINFLUSSUNG DES METHANOLSTOFFWECHSELS DURCH ÄTHANOL UND TOLBUTAMID. [Different influence of ethanol and tolbutamide on methanol metabolism].

Naunyn-Schmiedebergs Archiv für Pharmakologie (Berlin), 264(3): 298-299 (0 ref.), 1969.
G – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – liver, kidney – metab. proc. – alcohols – *CAAAL-14370 B-1018.

The effects of ethanol on methanol metabolism, and the actions of tolbutamide and amethopterin on the elimination of methanol, were studied in rats and dogs. In dogs which were poisoned by a single iv dose of 2 g/kg methanol and then given enough ethanol to maintain a plasma level of more than 0.5°/oo, the accumulation of formic acid in plasma was suppressed, and methanol elimination was only slightly impeded. In rats given 1 dose of 4 g/kg methanol iv, plus ethanol in sufficient amounts to maintain a plasma level of 1-2°/oo, the methanol remained in the organism twice as long. The question whether the self-induction of methanol is influenced by ethanol was investigated in young female Wistar rats (30-50 g). 2 groups received 1 dose of 4 g/kg methanol iv; in addition, 1 group received sufficient 30% ethanol po to achieve a plasma level of 1-2°/oo. In the ethanol group, the activity of the methanol-metabolizing system increased by about 120% until 72 hr after administration, whereas the enzyme system in the methanol-only group increased by 50% until 36 hr. It is concluded that ethanol cannot inhibit the self-induction of, nor stimulate, methanol metabolism.

1126. Rinkel, M., and Myerson, A.

EFFECT OF AMPHETAMINE SULFATE AND ALLIED DRUGS ON THE ALCOHOL LEVEL OF THE BLOOD.

A.M.A. Archives of Neurology and Psychiatry (Chicago), 45: 898-899 (1 ref.), 1941.
E – abst. – exp. cont. – exp. comp. – DC (decrease) – blood lev. – absorp., distrib., stor. – CNS – G.I. tract – amphetamines – autonomic agents – *CAAAL-3235-A2 A-1053.

Amphetamine sulphate, 30-40 mg iv or 20-40 mg po, depressed the blood alcohol concentration in man and animals. When the amphetamine was protected against quick absorption, the depressing effect was observed 72 hr after administration. The same result to a lesser degree, in order of declining effectiveness, was obtained with hydroxyamphetamine, epinephrine, and atropine. It was established that the inhibition of the alcohol concentration in the blood was due solely to decreased alcohol absorption from the gastrointestinal tract. In another experiment, pigeons were administered 3 g/kg alcohol by stomach tube, with or without 1 mg amphetamine. The pigeon receiving alcohol alone collapsed within 25 min, and the pigeon with amphetamine did not appear affected; 4 hr later, the effect was reversed—the former pigeon recovered and the latter collapsed. The drugs studied do not antagonize alcohol, but merely delay its absorption.

1127. Rinkel, M., and Myerson, A.

PHARMACOLOGICAL STUDIES IN EXPERIMENTAL ALCOHOLISM: 1. THE EFFECT OF SYMPATHOMIMETIC SUBSTANCES ON THE BLOOD-ALCOHOL LEVEL IN MAN.

J. Pharmacol. Exp. Ther. (Baltimore), 71: 75-86 (13 ref.), 1941.
E – exp. cont. – exp. comp. – DC (decrease) – psychot. humans – acute admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – cardiovasc. – G.I. tract – metab. proc. – respir. – amphetamines – autonomic agents – *CAAAL-2947-A1 A-1054.

14 schizophrenics and 1 chronic alcoholic (now abstinent) received 0.5-0.66 g/kg ethanol by different routes. Tested were the effects on the blood alcohol concentration of: amphetamine sulphate (20-40 mg); paredrine (10-40 mg); adrenaline chloride (0.1-2 mg); and atropine sulphate (2.6 mg or 1.3 mg) combined with amphetamine sulphate (20 mg); all drugs by different methods of administration. These drugs lowered the concentration of blood alcohol, probably by inhibiting the absorption from the alimentary tract, and by delaying the emptying time of the stomach. Quantitatively, amphetamine sulphate was the most effective, followed by paredrine, adrenaline chloride, and atropine sulphate.

1128. Rinkel, M., and Myerson, A.

ALCOHOL ABSORPTION AND INTOXICATION: THEIR MODIFICATION BY AUTONOMIC DRUGS.

Amer. J. Psychiat. (Hanover, N.H.), 98: 767-769 (0 ref.), 1942.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – cardiovasc. – G.I. tract – amphetamines – autonomic agents – sed., hypnot. – *CAAAL-3365-A1 A-1055.

The effects of amphetamine sulphate, paredrine, epinephrine, atropine sulphate, acetylcholine, acetyl-beta-methyl-choline (mecholy), carbaminoylcholine (doryl), and prostigmine on alcohol absorption and intoxication were studied in humans and pigeons. In experiments on humans, the sympathomimetic agents (no doses given), administered iv 1 min after 0.5-0.7 g/kg alcohol po, delayed absorption of alcohol from the alimentary tract and postponed intoxication; the relative order of effectiveness was: amphetamine, paredrine, adrenaline, and atropine. Tests on pigeons confirmed this effect. When the parasympathomimetic drugs were administered after the introduction of alcohol directly into the duodenum (but not after alcohol po), enhanced absorption was obtained with mecholy and doryl. When acetylcholine was given rectally 1-2 min before alcohol, alcohol absorption from the colon was enhanced. It is concluded that the use of these drugs in the treatment of alcoholism, with the exception of amphetamine sulphate in chronic alcoholism, cannot be recommended without reservation.

1129. Ris, F.

ALCOHOL UND CHLOROFORM. [Alcohol and chloroform].

Internationale Monatsschrift zur Erforschung des Alkoholismus und Bekämpfung der Trinksitten (Lausanne), 13: 318-320 (0 ref.), 1903.
 G – general – cross-tol. – humans – psychot. humans – CNS – anesthetics – *CAAAL-0 A-1056.

The author relates his experiences with chloroform narcosis. He observed that women, children, and psychiatric patients were much more easily anesthetized than adult men. The sole reason for this is alcohol abstinence. The chloroform narcosis is, according to the author, an infallible test whether the person under anesthesia uses alcohol, even in moderate quantities. Since this "alcohol reaction" in chloroform narcosis also occurs when the patient has been abstinent for some time, the author hypothesizes that some permanent, or at least long-lasting, change in the brain may be caused by alcohol consumption.

1130. Roach, M. K., Reese, W. N., and Creaven, P. J.

MICROSOMAL ETHANOL METABOLISM IN RAT LIVER.

Fed. Proc. (Bethesda), 28(2): 546 (2 ref.), 1969.
 E – abst. – exp. comp. – cross-tol. – mammals – in vitro – liver, kidney – barbiturates – *CAAAL-0 B-0433.

The properties of a hepatic microsomal mixed-function oxidase system were studied in rat liver microsomes. The activity of the system was increased by pretreatment with ethanol, but not by pretreatment with other known inductors. Pretreatment of animals with phenobarbital and benzo-

(alpha)-pyrene produced, respectively, a 100% and a 70% increase in activity. The results indicate that the microsomal ethanol-metabolizing system resembles other drug-metabolizing enzyme systems, in being susceptible to induction by phenobarbital and benzo-(alpha)-pyrene.

1131. Roach, M. K., Reese, W. N., Jr., and Creaven, P. J.
ETHANOL OXIDATION IN THE MICROSOMAL FRACTION OF RAT LIVER.
 Biochem. Biophys. Res. Commun. (New York), 36(4): 596-602 (10 ref.), 1969.
 E – exp. cont. – exp. comp. – DC (decrease) – mammals – in vivo – in vitro – metab. proc. – indust. intox. – miscellaneous – *CAAAL-0 B-0553.

To determine the nature of enzymatic ethanol oxidation, the control (complete system) was prepared by isolating a microsomal rat liver fraction and incubating it with ethanol, NADPH, and buffer. The acetaldehyde assayed by gas chromatography indicated the % of ethanol oxidation, which for the control was 100%. By analysis of test solutions, ethanol oxidation was seen to be NADPH- and oxygen-dependent, inhibited by carbon monoxide, sodium azide, and sodium cyanide, unaffected by SKF-525-A, and inhibited less than 50% by pretreatment with aminotriazole. The ethanol-oxidizing activity has been attributed to the microsomal mixed-function oxidase system, and it was also determined that NADPH could be replaced by a hydrogen peroxide-generating system, such as glucose plus glucose oxidase. Aminotriazole inhibited both the NADPH and the glucose plus glucose oxidase-dependent ethanol-oxidizing activities only by 40-50%, while the specific inhibition of catalase activity was significantly greater, thus indicating that 60% of the activity of the hydrogen peroxide-dependent microsomal oxidation of ethanol was due to catalase, and about 40% due to some other system.

1132. Robbins, B. H.
THE ABSORPTION, DISTRIBUTION AND EXCRETION OF CARBON TETRACHLORIDE IN DOGS UNDER VARIOUS CONDITIONS.
 J. Pharmacol. Exp. Ther. (Baltimore), 37(2): 203-216 (10 ref.), 1929.
 E – SEC – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – absorp., distrib., stor. – G.I. tract – respir. – anti-infectants – *CAAAL-0 A-1057.

5 dogs were given 10 or 50 cc of carbon tetrachloride (CCl_4) by injection into the intestine, plus 40 cc alcohol. There was a definite increase in the exhaled CCl_4 . Previous work had established that alcohol can change the CCl_4 toxicity when it is taken long before the CCl_4 , and, as the combined effect of the substances is entirely out of proportion to the CCl_4 dose, it is considered that alcohol affects the toxicity in some way other than by changing the rate of absorption from the intestinal tract.

1133. Robert, and Dervillée
L'INTOXICATION PROFESSIONNELLE PAR L'ALCOOL DANS L'INDUSTRIE DES POUDRES. [Occupational alcohol poisoning in the gunpowder industry].
 Ann. Med. Leg. (Paris), 28: 341-345 (1 ref.), 1948.
 F – general – DC (add., infra-add., unspec. incr.) – humans – cardiovasc. – CNS – G.I. tract – liver, kidney – senses – anesthetics – *CAAAL-5203-H69 A-1058.

The process of manufacture of gunpowder is described—considerable quantities of ethanol are used in this process. The mixture of ethanol and ethyl ether emits a vapour more toxic than either substance alone, and the toxicity of this mixture increases as the temperature is increased. Although ethanol is far less toxic in combination with ether than with amyl alcohol, which was formerly used and which led to severe poisonings, the effects are still substantial. In workers who had spent 10 or 15 yr in this occupation, the authors found nephritis with extensive albuminuria, the presence of casts and red blood corpuscles in the urine, and uremia (often exceeding 0.75 g). The poisoning from exposure to such a hazardous combination of substances assumes the status of a veritable occupational disease, which, however, is not officially recognized.

1134. Roberts, C.

TREATMENT OF ALCOHOLISM BY NUX VOMICA.

Brit. Med. J. (London), 1: 242-243 (0 ref.),

1888.

E – general – DC (antidotal) – humans – CNS – G.I. tract – anti-infectants – stimulants – *CAAAL-0
A-1059.

Nux vomica has been used for some time in the treatment of drunkenness. Full doses of the tincture of nux vomica, combined with rhubarb, soda, and full doses of carbonate of ammonia have been used with great success, and the author has satisfactorily employed nux vomica in combination with alkaline sol of bismuth, hydrocyanic acid, and carbonate of ammonia for the more acute cases, or combined with acid sol of strychnine plus iron and quinine in chronic cases. The author has had disappointing results with the few cases in which sc strychnine was used, and he rejects the opinion that strychnine is a true antidote to alcohol.

1135. Rodman, H.

REPORT OF A CASE OF CARBOLIC-ACID POISONING SUCCESSFULLY TREATED WITH ALCOHOL.

Medical Record (New York), 58(2): 70-72 (0 ref.),

1900.

E – general – case hist. – DC (antidotal) – humans – cardiovasc. – G.I. tract – liver, kidney – anesthetics – stimulants – *CAAAL-0
A-1060.

Reported is a case of phenol poisoning that was successfully treated with alcohol. A 60 yr-old woman swallowed 2 oz of pure carbolic acid by mistake. When the author reached the patient, she was unconscious; the pulse was imperceptible, the face was congested, there was extreme dyspnoea, the lips were cyanotic, the conjunctival and pupillary reflexes were absent, and the extremities were limp and cold. 4 oz of pure alcohol were administered by stomach tube; the stomach was drained 3 min later and washed with dilute alcohol. In the next 2 hr, strychnine and other cardiac stimulants were employed, and whiskey was injected sc at frequent intervals. In 5 days, the patient was fully recovered.

1136. Røe, O.

CLINICAL INVESTIGATIONS OF METHYL ALCOHOL POISONING WITH SPECIAL REFERENCE TO THE PATHOGENESIS AND TREATMENT OF AMBLYOPIA.

Acta Med. Scand. (Stockholm), 113(6): 558-608 (57 ref.),

1943.

E – general – case hist. – DC (antidotal) – humans – drug-dep. humans – blood lev. – absorp., distrib., stor. – acid-base, blood pH, elect. – cardiovasc. – CNS – G.I. tract – metab. proc. – respir. – senses – skel., muscle, skin – alcohols – *CAAAL-4176-A3
A-1061.

16 cases of methanol poisoning are described. It was observed that those patients who had drunk ethanol in addition to the methanol showed less severe symptoms than others who had not drunk ethanol. It is considered that ethanol possesses the capacity to displace methanol from the inner surfaces of cells, thereby checking the oxidation of the latter. Therefore, a milder course is given to the poisoning if ethanol is consumed just before or, better still, just after the drinking of methanol, the oxidation of which is then checked. All signs of poisoning may be averted if ethanol is drunk repeatedly during the first few days after methanol consumption, even if the latter has been taken in large quantities. Other recommended treatment includes the correction of acidosis by iv isotonic (1.3%) sodium bicarbonate sol, liberal flushing with fluid, protection from exposure to light, avoidance of all metabolic stimulants, exercise, hot baths, and thyroid extract.

1137. Røe, O.

METHANOL POISONING: ITS CLINICAL COURSE, PATHOGENESIS, AND TREATMENT.

Dissertation, University of Oslo, Norway, 253 pp. (100 ref.),

1946.

E – stat. surv. – case hist. – DC (antidotal) – post-mort. – humans – drug-dep. humans – blood lev.

– other drug lev. – absorp., distrib., stor. – acid-base, blood pH, elect. – blood comp., sites, lymph – cardiovasc. – CNS – G.I. tract – liver, kidney – metab. proc. – nerv. syst. – respir. – senses – skel., muscle, skin – alcohols – *CAAAL-4632-C3 A-1349.

This thesis (also published in *Acta Med. Scand.*, 126 (Suppl. 182), 1946) reviews the earlier literature on methanol poisoning and its treatment. There is agreement on the clinical picture, but disagreement as to the pathogenesis. 82 cases of acute methanol poisoning are reviewed, and the significance of acidosis is discussed. An important finding was the considerably longer latent period between ingestion and onset of symptoms in persons who had ingested ethanol as well as methanol. It is considered that ethanol prevents the absorption of methanol, and thus forestalls the conversion of methanol to formic acid. If the ethanol is consumed after the methanol, but before the appearance of symptoms, the latent period can be prolonged by an interval longer than that required for the elimination of the ethanol. This is because the production of formic acid ceases immediately upon the displacement of methanol from the respiratory enzyme; some elimination of formic acid from the body will still take place, however, resulting in a diminished inhibition of cellular respiration. The organic acids which have displaced bicarbonate will be partially oxidized, a certain amount of alkali will be set free, and the acidosis will be diminished. On the other hand, if ethanol is consumed at the same time, an increase of the alkali reserve will be impossible, and the latent period will only equal that necessary for ethanol oxidation. Due to the rapid oxidation of ethanol, a single dose will not prevent methanol poisoning, but, if the patient remains under the influence of ethanol for the first 3 or 4 days after methanol intake, he will remain symptom-free, even if he has drunk several hundred ml of methanol.

1138. Røe, O.

METHANOL POISONING: ITS CLINICAL COURSE, PATHOGENESIS AND TREATMENT.

Acta Med. Scand. (Stockholm), 126(Suppl. 182): 253 pp. (100 ref.), 1946.
E – stat. surv. – case hist. – DC (antidotal) – DC (decrease) – post-mort. – humans – drug-dep. humans – blood lev. – other drug lev. – absorp., distrib., stor. – acid-base, blood pH, elect. – blood comp., sites, lymph – cardiovasc. – CNS – G.I. tract – liver, kidney – metab. proc. – nerv. syst. – respir. – senses – skel., muscle, skin – alcohols – *CAAAL-4632-C3 A-1062.

This article (also published as a thesis) reviews the earlier literature on methanol poisoning and its treatment. There is agreement on the clinical picture, but disagreement as to the pathogenesis. 82 cases of acute methanol poisoning are reviewed, and the significance of acidosis is discussed. A significant finding was the considerably longer latent period between ingestion and onset of symptoms in persons who had ingested ethanol as well as methanol. It is considered that ethanol prevents the absorption of methanol, and thus forestalls the conversion of methanol to formic acid. If the ethanol is consumed after the methanol, but before the appearance of symptoms, the latent period can be prolonged by an interval longer than that required for the elimination of the ethanol. This is because the production of formic acid ceases immediately upon the displacement of methanol from the respiratory enzyme; some elimination of formic acid from the body will still take place, however, resulting in a diminished inhibition of cellular respiration. The organic acids which have displaced bicarbonate will be partially oxidized, a certain amount of alkali will be set free, and the acidosis will be diminished. On the other hand, if ethanol is consumed at the same time, an increase of the alkali reserve will be impossible, and the latent period will only equal that necessary for ethanol oxidation. Due to the rapid oxidation of ethanol, a single dose will not prevent methanol poisoning, but, if the patient remains under the influence of ethanol for the first 3 or 4 days after methanol intake, he will remain symptom-free, even if he has drunk several hundred ml of methanol.

1139. Røe, O.

METHANOL POISONING.

Bull. Schweiz. Akad. Med. Wiss. (Basel), 3: 204-210 (0 ref.), 1947.
E – FS – GS – IS – stat. surv. – DC (antidotal) – DC (decrease) – humans – blood lev. – acid-base,

blood pH, elect. – CNS – metab. proc. – senses – skel., muscle, skin – alcohols – *CAAAL-5291-E4
A-1063.

Clinical investigations of methanol poisoning in Norway have shown that the outcome of poisoning depends entirely on the degree of acidosis. Ethyl alcohol acts as a powerful antidote, since its surface activity is greater than that of methanol, and it can displace the latter from the respiratory ferment, thereby preventing the oxidation of methanol to formic acid. In postmortem examinations, very pronounced degenerative changes are to be found in ganglion cells of the retina. The author has not found similar changes in the retina of experimental animals, which do not develop a marked acidosis. Thus, there are fundamental differences in the action of methanol on human beings and on animals.

1140. Røe, O.

METANOLFORGIFTNING. [Methanol poisoning].

T. Norsk. Laegeforen. (Oslo), 68: 572-574 (0 ref.),

1948.

N – general – DC (antidotal) – humans – blood lev. – acid-base, blood pH, elect. – senses – alcohols – *CAAAL-5898-N8
A-1064.

In methanol poisoning, the latent period of the poisoning is about 18 hr, during which the patient gives little impression of being drunk. The symptoms are severe acidosis, combined with bilateral amblyopia or amaurosis. Ethanol prevents acidosis, but its action is transient, and the elimination of methanol is slow. When taken simultaneously with methanol, it prolongs the latent period, and thus may obscure the diagnosis. Excessive ethanol treatment is not advised—a blood alcohol concentration of 1°/oo (100 ml brandy or gin, followed by 20 ml doses/hr) is sufficient.

1141. Røe, O.

THE ROLES OF ALKALINE SALTS AND ETHYL ALCOHOL IN THE TREATMENT OF METHANOL POISONING.

Quart. J. Stud. Alcohol (New Haven), 11(1): 107-112 (12 ref.),

1950.

E – general – case hist. – DC (antidotal) – humans – blood lev. – other drug lev. – absorp., distrib., stor. – acid-base, blood pH, elect. – metab. proc. – senses – alcohols – *CAAAL-4499-N8

A-1065.

The author defends his work against the alleged inaccurate descriptions of it by Agner, Kjell et al. (Acta Physiol. Scand. (Stockholm), 13: 87-94, 1947; Svenska Läkartidningen (Stockholm), 45: 995-999, 1948; and Quart. J. Stud. Alcohol (New Haven), 9(4): 515-522, 1949), and recapitulates the results of his own investigations. Agner and co-workers failed to give the recommended treatment, and their patients were in a hopeless state prior to ethanol treatment. Treatment with ethanol is a supplementary measure which, if carried out properly, will prevent the recurrence of acidosis. If the alkali reserve is low, bicarbonate must be given in the first instance. Ethanol treatment should be particularly considered when a lengthy transportation of the patient renders prompt and adequate treatment with bicarbonate difficult or impossible. The author has several times seen patients who had begun a trip to the hospital symptom-free, and arrived blind or dead.

1142. Røe, O.

THE METABOLISM AND TOXICITY OF METHANOL.

Pharmacol. Rev. (Baltimore), 7: 399-412 (54 ref.),

1955.

E – general – review – case hist. – DC (antidotal) – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – in vitro – blood lev. – species or sex diff. – acid-base, blood pH, elect. – CNS – liver, kidney – metab. proc. – senses – alcohols – elect., water-bal. agents – *CAAAL-6075-A4
A-1066.

A comprehensive review is presented concerning the oxidation and elimination of methyl alcohol, the production of intermediary substances, and the mechanisms involved. The symptoms of methyl

alcohol poisoning, particularly the acidosis and the involvement of the eyes, are described, and the pathology, development of the poisoning, and treatment are outlined. Ethanol inhibits the metabolism of methanol to toxic products, and may be administered to good effect to prevent the recurrence of acidosis. A concentration of 100 mg/100 ml of blood is sufficient, particularly in view of the finding that a concentration of 46 mg% can decrease the rate of methanol metabolism by 72%. It must be given often enough to keep the ethanol concentration fairly constant. No beneficial effect of ethanol can be expected in animal experiments, because animals do not develop acidosis, even when methanol oxidation is allowed to proceed at a normal rate, and it is thus not surprising that Gilger et al. (Amer. J. Ophthal., 35(5, part 2): 113-126, 1952) found that ethanol increased the toxicity of methanol.

1143. Røe, O.

METANOLS TOKSISITET. [Toxicity of methanol].

Nord. Med. (Stockholm), 54(41): 1549-1551 (19 ref.),

1955.

S – ES – general – DC (antidotal) – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – in vitro – species or sex diff. – acid-base, blood pH, elect. – metab. proc. – alcohols – *CAAAL-7442-N8 A-1067.

Prompted by the results of experiments of Moeschlin and Garson (Schweiz. Med. Wschr. (Basel), 85: 61-62, 1955) and others on methanol poisoning in animals, the author stresses the fact that there is a fundamental difference in the toxic action of methanol on animals and human beings. The severe acidosis, which is the essential and all-important symptom in human methanol poisoning, does not exist in the animals used for these experiments (i.e., non-primates). Alkali and ethyl alcohol administered to poisoned animals are further toxic agents in addition to the lethal doses of methanol (10-12 g/kg) commonly used in such experiments. In human beings, both substances help to combat acidosis—the alkali by substituting the loss of bicarbonate, and the ethyl alcohol by reducing or even preventing the oxidation of methanol to toxic products.

1144. Røe, O.

ÜBER DEN WERT DER TIEREXPERIMENTELLEN UNTERSUCHUNGEN FÜR DAS STUDIUM DER TOXIZITÄT DES METHANOLS: BEMERKUNGEN ZU DER ARBEIT VON S. MOESCHLIN UND H. GARSON: „UNTERSUCHUNGEN ÜBER DIE ÄTHYLALKOHOLTHERAPIE DER EXPERIMENTELLEN METHYLALKOHOLVERGIFTUNG“.

[The value of animal experimentation for the study of toxicity of methanol: Remarks concerning the work of S. Moeschlin and H. Garson: “Study of the ethyl alcohol treatment of experimental methyl alcohol poisoning”].

Schweiz. Med. Wschr. (Basel), 85(34): 813-815 (19 ref.),

1955.

G – ES – general – DC (antidotal) – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – in vitro – species or sex diff. – acid-base, blood pH, elect. – metab. proc. – alcohols – *CAAAL-7442-N8 A-1068.

Prompted by the results of experiments of Moeschlin and Garson (Schweiz. Med. Wschr. (Basel), 85: 61-62, 1955) and others on methanol poisoning in animals, the author stresses the fact there is a fundamental difference in the toxic action of methanol on animals and human beings. The severe acidosis, which is the essential and all-important symptom in human methanol poisoning, does not exist in the animals used for these experiments (i.e., non-primates). Alkali and ethyl alcohol administered to poisoned animals are further toxic agents in addition to the lethal doses of methanol (10-12 g/kg) commonly used in such experiments. In human beings, both substances help to combat acidosis—the alkali by substituting the loss of bicarbonate, and the ethyl alcohol by reducing or even preventing the oxidation of methanol to toxic products.

1145. Rohde, H.

ÜBER DEN EINFLUSS VON NATRIUM BICARBONICUM AUF DEN VERLAUF DER ALIMENTÄR-ALKOHOLÄMISCHEN KURVE BEIM MENSCHEN. [The effect of sodium

bicarbonate on the course of the alimentary blood alcohol curve in humans].

Dissertation, Center for Studies on Pathological Physiology, Friedrich Wilhelms University, Berlin, Germany, 22 pp. (40 ref.), 1938.

G – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – metab. proc. – elect., water-bal. agents – *CAAAL-0 A-1350.

The effects of sodium bicarbonate (SB) on the blood alcohol level were investigated in 7 healthy humans. On 1 occasion, the subjects drank 25 cc absolute alcohol in 400 cc water. After a 4-6 day interval (individual dose administration), a sol containing the same amount of alcohol plus 6 g SB was drunk. Blood tests were performed on each occasion (Widmark method). It was found that, after alcohol alone, the BAL was significantly high after 2 hr in 2 cases, and after 2 1/2 hr in 5. After alcohol plus SB, the BAL returned to normal after 1 1/2 hr. In some cases (4 out of 7), after alcohol plus SB, a slightly higher BAL was noticed after the first half hr (and, in 1 case, after the first hr), but after this time the BAL declined rapidly, so that in all subjects no alcohol could be detected at the end of the second hr. It is concluded that SB, at least in humans, appears to promote alcohol absorption and metabolism, insofar as this can be detected by the Widmark method.

1146. Rollins R. L., Jr., and Cefalu, S. J.

BROMIDE INGESTION IN ALCOHOLICS.

N. Carolina Med. J. (Winston-Salem), 29: 342-343 (5 ref.),

1968.

E – SEC – general – conj. addict. – drug-dep. humans – blood lev. – *CAAAL-0

B-0434.

A study was made on alcoholic and non-alcoholic patients to determine the incidence of bromides in the blood of these patients. The results of tests in 100 male and female patients consecutively admitted to hospitals in several North Carolina counties were negative, whereas in 2 series of tests involving 100 male alcoholic patients/series, positive results were found in 7 and 5% of the cases, respectively. Symptoms and treatment for bromide poisoning are discussed. 2 case histories are cited. The study indicates that alcoholic patients have a higher incidence of bromide ingestion than other psychiatric patients; also, acute alcoholism and bromide intoxication may co-exist.

1147. Röseler, P.

BEOBSACHTUNGEN UND UNTERSUCHUNGEN ÜBER DIE GEMEINSAME WIRKUNG VON ALKOHOL UND KOHLENMONOXYD. [Observations and investigations on the interaction of alcohol and carbon monoxide].

Dissertation, Faculty of Medicine of the Free University of Berlin, West Germany, 61 pp. (92 ref.), 1961.

G – exp. cont. – DC (add., infra-add., unspec. incr.) – post-mort. – humans – mammals – acute admin. – in vivo – dose resp. – blood lev. – indust. intox. – *CAAAL-0 A-1069.

Results of preliminary in vivo investigations on 65 mice for the mean carbon monoxide (CO)-haemoglobin (Hb) concentration at lethal intoxication levels, gave a value of $68 \pm 6\%$ CO-Hb. In experiments on alcohol tolerance with 6,000-10,000 mg/kg alcohol, 50% of the animals survived. The combined application of CO plus alcohol was studied in a series of 3 tests in which alcohol was administered in doses of $7,730 \pm 878$ mg/kg, $3,996 \pm 302$ mg/kg, and $2,775 \pm 345$ mg/kg, respectively. The lethal CO-Hb concentrations in the 3 series of tests were $52 \pm 4\%$, $56 \pm 4\%$, and $59 \pm 4\%$, respectively. The degree of regression was determined by means of statistical calculations. A significant decrease in the lethal CO-Hb concentration, as a function of increased alcohol doses, was noted. The lethal CO-Hb concentration in man is $66 \pm 6\%$ CO-Hb, and the lethal alcohol blood level $3.5^\circ/\text{oo}$. In 4 post-mortem findings, the patients died at a CO-Hb concentration of 45 to 48.5%; the blood alcohol level was 1.5 to $2.7^\circ/\text{oo}$. Synergism was therefore observed. The question whether the effect is additive or potentiative would need further investigation.

1148. Rosenbaum, M.

ADAPTATION OF THE CENTRAL NERVOUS SYSTEM TO VARYING CONCENTRATIONS OF ALCOHOL IN THE BLOOD.

A.M.A. Archives of Neurology and Psychiatry (Chicago), 48: 1010-1012 (5 ref.), 1942.
 E – exp. – DC (decrease) – drug-dep. humans – acute admin. – in vivo – blood lev. – CNS – stimulants
 – *CAAAL-3769-A1 A-1070.

8 chronic alcoholics received 10 g/kg ethanol (30-50% sol) po. 1-2 hr later, 0.25-0.5 g/kg alcohol was given. The blood alcohol levels at which the subjects became sober or drunk were determined; in all experiments, the subjects became intoxicated at a lower level than that at which they became sober. 2 patients were given large alcohol doses to make them comatose, and then they received 9 cc 10% metrazol sol. In a few min, the patients became conscious and responsive, despite the fact that there was no decrease in the blood alcohol level. In 1 patient, the amount of alcohol in the blood preceding the metrazol was 428 mg/100 cc, and, 11 min later, when he was out of the coma after metrazol administration, the alcohol level was 439 mg/100 cc. It is concluded that the blood alcohol concentration is a poor indicator of the degree of intoxication.

1149. Rosenfeld, G.

POTENTIATION OF THE NARCOTIC ACTION AND ACUTE TOXICITY OF ALCOHOL BY PRIMARY AROMATIC MONOAMINES.

Quart. J. Stud. Alcohol (New Haven), 21(4): 584-596 (16 ref.), 1960.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – other drug lev. – cardiovasc. – CNS – metab. proc. – respir. – amphetamines – autonomic agents – cardiovasc. agents – *CAAAL-9432-B2 A-1071.

A study was conducted of the possible interaction of several primary aromatic monoamines normally found in the brain with alcohol when both are administered to groups of mice. When 0.91 mM/kg of serotonin, dopamine, tyramine, and gamma-aminobutyric acid were administered 30 min before 4.5 g/kg ethanol (a sublethal hypnotic dose), there were no deaths. However, if the same dose of serotonin, tryptamine, or dopamine was given 30 min after the same dose of alcohol, mortalities of 83%, 51%, and 50%, respectively, were the result. In addition, a group of mice was injected with 0.23 mM of amphetamine per kg 30 min after 4.5 g/kg ethanol. The mice exhibited a mean sleeping time of only 59 min, compared to the control mean of 74 min. Thus, despite the chemical similarity to dopamine and tyramine, amphetamine behaves quite differently with respect to alcohol.

1150. Rosenthal, S. M.

SOME EFFECTS OF ALCOHOL UPON THE NORMAL AND DAMAGED LIVER.

J. Pharmacol. Exp. Ther. (Baltimore), 38: 291-301 (9 ref.), 1930.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – CNS – G.I. tract – glands – liver, kidney – anesthetics – anti-infectants – *CAAAL-2938-B2 A-1072.

Alcohol administered po to normal dogs in doses of 1 cc/kg did not impair normal liver function. The results were similar in dogs in which the livers had been damaged with chloroform or carbon tetrachloride. Despite these results, alcohol did greatly increase the susceptibility of the dogs to chloroform anesthesia (by inhalation), even after the visible effects of alcohol had worn off. 6 out of 10 dogs which had received alcohol (2 cc/kg po) 3 to 4 hr prior to the anesthetic died, whereas those which did not receive the alcohol all survived.

1151. Roth, G. M., and Sheard, C.

THE EFFECT OF SMOKING ON THE VASODILATATION PRODUCED BY THE ORAL ADMINISTRATION OF ALCOHOL.

Central Society for Clinical Research, Proceedings (Chicago), 16: 60-61 (0 ref.), 1943.
 E – exp. cont. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – cardiovasc.
 – skel., muscle, skin – *CAAAL-3990-D1 A-1073.

In 35 normal persons, the skin temperatures of the extremities, the blood pressure, and the pulse rate were simultaneously measured after smoking 2 cigarettes, both with and without a dose of 95% alcohol. The alcohol did not significantly alter vasoconstriction produced by smoking. At the height of the vasodilation from alcohol, the blood pressure rise ranged from 4-50 mm mercury (systolic) and from 3 to 24 mm mercury (diastolic). In most instances in which the blood pressure showed the least change, the rise in pulse rate was greatest. Skin temperatures decreased from 1 to 6.5°C in the toes, and from 1 to 4°C in the fingers.

1152. Roth, G. M., and Sheard, C.
 THE EFFECT OF SMOKING ON THE VASODILATATION PRODUCED BY THE ORAL
 ADMINISTRATION OF 95 PER CENT ETHYL ALCOHOL OR A SUBSTANTIAL MEAL.
 Amer. Heart J. (St. Louis), 33: 654-662 (8 ref.), 1947.
 E – exp. cont. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – cardiovasc.
 – skel., muscle, skin – *CAAAL-4769-D1 A-1074.

65 normal subjects smoked 2/3 of each of 2 cigarettes in succession, both with and without 30 cc 95% ethanol. Skin temperature of the toes and fingers, blood pressure, and pulse rate were all determined simultaneously. After smoking and alcohol intake, the average skin temperature of the toes returned to the basal level, and there was a decrease of only 1.3°C in the skin temperature of the fingers. Although this appears to indicate that alcohol prevented the vasoconstriction produced by smoking, the skin temperature of the toes was lower than the basal level in 72% of the subjects when smoking was done after alcohol ingestion. The decrease after alcohol was of about the same magnitude as without alcohol. The differences in blood pressure and pulse rate were not significant.

1153. Roth, J. A., and Ivy, A. C.
 THE SYNERGISTIC EFFECT OF CAFFEINE UPON HISTAMINE IN RELATION TO
 GASTRIC SECRETION.
 Amer. J. Physiol. (Bethesda), 142: 107-113 (6 ref.), 1944.
 E – exp. cont. – DC (supra-add. incr.) – humans – mammals – acute admin. – in vivo – cardiovasc.
 – G.I. tract – stimulants – *CAAAL-4172-B1 A-1075.

Experiments were conducted on cats and humans with histamine and caffeine. To test whether a synergistic relation exists between alcohol and caffeine, 5 human subjects received 50 cc 7% alcohol, 250 mg caffeine (as sodium benzoate in 50 cc water), or 250 mg caffeine in 50 cc 7% alcohol, and the gastric secretory response was plotted. The response to caffeine-alcohol was, on the average, 65.9% (calculated on the mean responses) greater than the sum of the individual responses to alcohol and caffeine given separately for a similar 70-min period. The response to the combined dose was prolonged, and was maintained at a high level.

1154. Rouet, C.
 L'ANESTHÉSIE CHEZ L'ALCOOLIQUE. [Anesthesia in the alcoholic].
 Cah. Anesth. (Paris), 3(7): 629-642 (0 ref.), 1956.
 F – general – cross-tol. – drug-dep. humans – CNS – metab. proc. – anesthetics – autonomic agents
 – barbiturates – musculoskel. agents – sed., hypnot. – unclass. ther. agents – *CAAAL-0 A-1076.

The metabolism and excretion of alcohol are described. The treatment of alcoholics, particularly with reference to the administration of anesthesia, is discussed. One patient in a state of intoxication was

given a stomach lavage and then anesthetized with 50 cg pentothal. A second operation required considerable doses of pentothal, curare, and cyclopropane; nevertheless, the patient awakened on the operating table at the end of the operation. If time is available, the author prefers to try to stimulate the enzymatic processes which not only assure the catabolism of alcohol, but which intervene at all stages of metabolism and attack the metabolic products; for this, curethyl B and the vitamin B complex are useful. Otherwise, substances (gardenal, strychnine, and tranquilizers) relieving the nervous effects of alcohol can be used. With respect to the resistance of alcoholics to anesthesia, one of the possible methods of dealing with the problem is to administer alcohol po 2-3 hr prior to the operation; the author agrees that this can be done, or an iv perfusion of an alcohol serum (60 cc ethanol/1,000 cc) can be given 1 hr before the operation.

1155. Roy, P. B., Mercure, J., Lebel, G., and Bourque, R.
CONTRIBUTION À L'ÉTUDE DU "LIBRIUM" DANS LE TRAITEMENT DES
ALCOOLIQUES. [Contribution to the study of "librium" in the treatment of alcoholics].
Un. Med. Canada (Montreal), 92: 218-220 (2 ref.), 1963.
F – ES – SEC – general – DC (add., infra-add., unspec. incr.) – drug-dep. humans – CNS –
tranquilizers – *CAAAL-10465-N47 A-1077.

This article reports on the effects of methaminodiazepoxide upon 50 hospitalized alcoholic patients. The usual dose was 100-200 mg/day orally or 50-100 mg im for the agitated patient, and 50-100 mg iv for the severely agitated patient. Side effects were few. The drug was remarkably effective upon tension and anxiety, producing excellent results in 15 of 39 patients, good in 20, and poor in 4. It was observed that the drug may have a potentiating action on alcohol, and the authors note that such an interesting possible effect merits further and special investigation.

1156. Royer, R., Debry, G., and Lamarche, M.
RECHERCHES EXPÉRIMENTALES SUR LES RÉACTIONS VASOMOTRICES À
L'ALCOOL APRÈS ADMINISTRATION DE QUELQUES SULFAMIDES
HYPOGLYCÉMIANTS. [Experimental studies on vasomotor reactions to alcohol after
administration of some hypoglycemic sulfonamides].
Thérapie (Paris), 17: 989-997 (14 ref.), 1962.
F – ES – SpS – exp. cont. – exp. comp. – general – DC (sensit.) – humans – mammals – chronic admin.
– in vivo – cardiovasc. – anti-infectants – unclass. ther. agents – *CAAAL-0 A-1456.

The clinical symptoms and mechanism of vasomotor reactions to hypoglycemic sulfonamides, and the results of an experiment on rats are discussed. Of 160 patients receiving chlorpropamide, 16 experienced vasomotor reactions; in 13 of these, the reaction was precipitated by alcohol. The phenomenon, lasting about 1 hr, was characterized by an increased pulse rate and blood pressure, intense vasodilation of the face, and a feeling of warmth and reddening in the face, neck and other body areas. In groups of Wistar rats (200 g) chronically fed water and/or a 5% alcohol sol, the effects on alcohol consumption of the following conditions were studied: normal and deficient diet, disulfiram (15 mg/kg/day po for 5 days), chlorpropamide (100-500 mg/day po for 17 days), phenbutamide (100-300 mg/day po for 14 days), glybuthiazol (dosage unstated), and insulin (0.2 units/day sc). Alcohol consumption was increased in rats placed on a deficient diet, and was decreased in animals given disulfiram, chlorpropamide, or phenbutamide. Results were inconclusive with glybuthiazol, and no change in alcohol consumption was noted with chronic insulin administration. It is concluded that a number of hypoglycemic sulfonamides can provoke vasomotor reactions in 10-20% of the patients treated, and that these reactions are potentially dangerous in persons with cardiovascular disease. Although alcohol can precipitate or enhance the reaction, the phenomenon can also occur in the absence of alcohol, and, for this reason, it is not classed as a true disulfiram-alcohol reaction.

1157. Royer, R., and Lamarche, M.

ÉTUDE EXPÉRIMENTALE DE L'ACTIVITÉ ANTI-ALCOOL DE DIVERSES SUBSTANCES. [Experimental study of the anti-alcoholic action of various substances].

J. Physiol. (Paris), 56: 437-438 (1 ref.),

1964.

F – abst. – exp. comp. – DC (sensit.) – mammals – acute admin. – in vivo – psychol. perform. – cardiovasc. – nerv. syst. – respir. – alcohols – hormones, hormone antag. – unclass. ther. agents – *CAAAL-0

A-1314.

The authors outline a rational 2-step method for testing new “anti-alcoholic” substances. 1 part, detection, involves the administration of the substance to rats trained to prefer a 5% alcohol sol to water, to see if the rats reject the alcohol. A second part, pharmacodynamic reactions, involves force-feeding of the substance to rats 48 hr prior to the injection of alcohol; respiratory frequency, blood pressure, vasomotor activity, and heart electrical activity are measured. This method has been used by the authors to test various substances. Those found to be anti-alcoholic, or capable of inducing an intolerance reaction, were: disulfiram, unpurified animal charcoal, chlorpropamide, tolbutamide, and phenbutamide. Those which were not anti-alcoholic were: insulin, butyric alcohol, sulfanilamide, and purified animal charcoal. Free HS groups in the form of cysteine were found to accentuate the intolerance reaction.

1158. Royer, R., Debry, G., Lamarche, M., and Kissel, P.

SULFAMIDES HYPOGLYCÉMIANTS ET EFFET ANTABUSE. [Hypoglycemic sulphonamides and the antabuse effect].

Presse Med. (Paris), 72(12): 661-665 (55 ref.),

1964.

F – exp. cont. – case hist. – DC (sensit.) – humans – mammals – acute admin. – in vivo – cardiovasc. – respir. – hormones, hormone antag. – *CAAAL-11057-M3

A-1313.

Rats and rabbits pretreated with various hypoglycemic sulfonamides were given sc injections of alcohol. Respiration, pulse rate, arterial pressure, and pupil dilation were measured. Chlorpropamide- or tolbutamide-pretreated animals reacted with increased respiration rate and arterial pressure drop, but showed little change in pulse rate. Metahexamide did not produce these disturbances, and control animals given alcohol alone had only moderate respiratory frequency and amplitude. Dilation and constriction of arteries or pupils were not clearly defined with any of the above 3 drugs. Those drugs found capable of conditioning a rat to avoid an alcohol drink were, in order of effectiveness, phenbutamide, chlorpropamide, tolbutamide, and carbutamide. The drugs without this effect included metahexamide, the m-isomer of carbutamide, and glybuthiazol. Considerable discussion on the chemistry of these reactions is presented. In another experiment, ill alcoholics received 250 to 500 mg of chlorpropamide per morning, and were offered wine 3 times at 20 min intervals. 3 had no reaction, 8 reacted to each drink of wine, 5 reacted less than half the time, and 5 more than half the time. It is concluded that this and related drugs could be used to develop a distaste for alcohol.

1159. Royer, R., Lamarche, M., and Rombach, M.

RÔLE DE LA BRADYKININE DANS LA RÉACTION VASOMOTRICE MÉDICAMENT ALCOOL. [Role of bradykinin in the vasomotor drug-alcohol reaction].

J. Physiol. (Paris), 60: 536-537 (3 ref.),

1968.

F – SEC – abst. – exp. cont. – mammals – acute admin. – chronic admin. – in vivo – cardiovasc. – respir. – stimulants – unclass. ther. agents – *CAAAL-0

B-1019.

2 groups of 10 rabbits each, 1 group acting as control, and a test group having been pretreated with 100 mg disulfiram/kg/day for 3 days, were anesthetized with 30 mg/kg pentobarbital. Synthetic bradykinin was injected iv as a 1 µg/ml sol in single doses of 0.5, 1, 2, or 4 µg/3 kg, or continuously perfused iv as a 1, 2, or 4 µg/ml sol at a rate of 0.5, 1, or 2 ml/min, respectively. Respiration, arterial pressure, and electrocardiogram (ECG) effects were monitored. In controls given a single bradykinin injection, a hypotensive effect was evident at the 1 µg dose level; alteration in the respiratory rate

and amplitude was seen only with the highest dosages. The test (disulfiram) group showed no significant deviation from controls. In animals given continuous iv perfusions, there was again no significant difference between test and control animals. A hypotension, generally varying in proportion to the dosage, was observed, but there was no appreciable alteration in respiratory volume or rate. The ECG was not altered, except for a tachycardic reaction to the hypotension. It is concluded that bradykinin acts in a manner similar to that of serotonin, but with less intensity of hypotension, duration, and constancy; neither drug is potentiated by disulfiram. Bradykinin is therefore capable of playing the same role as serotonin in the vasomotor drug-alcohol reaction.

1160. Rozhnov, V. E., and Ratner, K. S.

VLIIANIE MALYKH DOZ ALKOGOLIA I EGO SOCHETANII S FENAMINOM I AMINAZINOM NA USLOVNUIU ZASHCHITNOMIGATEL'NUIU REAKTSIU PRI KHRONICHESKOM ALKOGLIZME. [Effect of small doses of alcohol and combinations of it with phenamine and aminazine on the conditioned defence-blink reaction in chronic alcoholism]. Zh. Nevropat. Psikhiat. Korsakov (Moscow), 67: 273-280 (42 ref.), 1967.
R – FS – exp. – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – mot. perform. – CNS – nerv. syst. – respir. – skel., muscle, skin – amphetamines – tranquilizers – *CAAAL-0 B-0435.

The administration of low doses of alcohol or alcohol combined with aminazine (50 mg im) to chronic alcoholics often depressed the motivating component of the conditioned defense-blink reaction (in comparison to the control). The stimulating effect (increased amplitude of eye movement) of the combination of alcohol with phenamine, 10 mg po, was more pronounced in the sick. Combinations of alcohol and phenamine and aminazine decreased movement of the vegetative components of the conditioned reaction more frequently in the ill than in the healthy. In chronic alcoholics, an isolated intake of alcohol sometimes manifested a tonic action on the vegetative functions.

1161. Rubin, E., and Lieber, C. S.

INCREASE OF HEPATIC DRUG METABOLIZING ENZYMES INDUCED BY ETHANOL. Fed. Proc. (Bethesda), 27(2): 605 (0 ref.), 1968.
E – abst. – exp. cont. – cross-tol. – mammals – chronic admin. – in vivo – liver, kidney – anesthetics – tranquilizers – *CAAAL-0 B-0436.

Because it was observed that many alcoholics are unusually tolerant of drugs such as anesthetics and tranquilizers, determinations were made in rats of the effect of chronic ethanol administration on hepatic drug-metabolizing enzymes, both in a complete diet and in one deficient in protein and lipotropes. After 2 weeks of isocaloric substitution of ethanol for carbohydrate (36% of total calories), hepatic lipids increased by 96% with the complete diet and by 133% with the deficient diet. Compared to pair-fed controls, there was an average 7.5-fold increase in aniline hydroxylase in the complete diet group, and a 13-fold increase in the deficient diet group. Nitroreductase was increased by 52% in the complete diet group and by 81% in the deficient diet group. It is concluded that a deficient diet, similar to that of many alcoholics, potentiates the stated effects.

1162. Rubin, E., Hutterer, F., and Lieber, C. S.

ETHANOL INCREASES HEPATIC SMOOTH ENDOPLASMIC RETICULUM AND DRUG-METABOLIZING ENZYMES. Science (Washington), 159(3821): 1469-1470 (13 ref.), 1968.
E – exp. cont. – cross-tol. – mammals – chronic admin. – in vivo – liver, kidney – metab. proc. – sed., hypnot. – *CAAAL-0 B-0437.

32 male rats were fed ethanol for 2 weeks, together with diets either adequate or deficient in protein and choline, the latter intake being similar to that of many alcoholics. Each rat fed ethanol was

matched with a similar control which received the appropriate diet without ethanol. Hepatic lipids, smooth endoplasmic reticulum, and the activities of drug-metabolizing enzymes (aniline hydroxylase and nitroreductase) were increased with the adequate diet, but were increased more appreciably with the deficient one. The authors speculate that these results may explain the increased tolerance of alcoholics to drugs such as sedatives.

1163. Rubin, E., and Lieber, C. S.

HEPATIC MICROSOMAL ENZYMES IN MAN AND RAT: INDUCTION AND INHIBITION BY ETHANOL.

Science (Washington), 162(3854): 690-691 (11 ref.),

1968.

E – exp. cont. – exp. comp. – cross-tol. – DC (unchanged) – mammals – acute admin. – chronic admin. – in vitro – liver, kidney – metab. proc. – barbiturates – *CAAAL-0 B-0438.

Tests were performed to determine the effect of ethanol on the activities of hepatic microsomal enzymes in humans and rats. The feeding of ethanol, which comprised 26% of the total calories in the diet, significantly increased the activities of hepatic pentobarbital and benzpyrene hydroxylases in rats, and, in humans on a 42% ethanol-calorie diet, doubled pentobarbital hydroxylase activity. In vitro ethanol inhibited aniline, pentobarbital, and benzpyrene hydroxylases. These data may explain, at least in part, the increased tolerance of alcoholics to sedatives when sober, and the enhanced sensitivity to sedatives when inebriated.

1164. Rubin, E., and Lieber, C. S.

ALCOHOL, OTHER DRUGS, AND THE LIVER.

Ann. Intern Med. (Philadelphia), 69(5): 1063-1067 (31 ref.),

1968.

E – exp. – review – cross-tol. – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – chronic admin. – in vivo – in vitro – CNS – liver, kidney – metab. proc. – barbiturates – sed., hypnot. – *CAAAL-0 B-0439.

The literature is reviewed with reference to experiments on the interaction of alcohol and barbiturates. Ethanol was administered for 12 days to human volunteers, and was found to result in a doubling of hepatic pentobarbital hydroxylase. These results may explain, at least partly, the resistance of chronic alcoholics to the action of sedatives. Conversely, the potentiation of the barbiturate effect in inebriated persons is explained by the observation that ethanol, in concentrations often found in the blood of intoxicated individuals, inhibits the in vitro activity of a variety of microsomal enzymes, including pentobarbital hydroxylase. It is also known that pretreatment with hexobarbital, a potent inducer of microsomal enzymes, accelerates the elimination of alcohol.

1165. Rubin, E., Bacchin, P., Gang, H., and Lieber, C. S.

INDUCTION AND INHIBITION OF HEPATIC MICROSOMAL AND MITOCHONDRIAL ENZYMES BY ETHANOL.

Lab. Invest. (New York), 22(6): 569-580 (36 ref.),

1970.

E – exp. cont. – cross-tol. – humans – mammals – acute admin. – chronic admin. – in vivo – CNS – liver, kidney – metab. proc. – *CAAAL-0 B-0581.

Acute and chronic tests were performed on rats and humans. 200 g Sprague-Dawley rats received for 15-24 days a nutritionally-adequate, a protein- and choline-deficient, or a low-fat diet, with or without ethanol as 36% of total cal. In another test, fasted rats received a single dose of 1.5 or 8 g/kg ethanol by gastric intubation. 3 healthy humans were fed for 12 days a diet in which ethanol comprised 42% of total cal. In in vitro tests, ethanol was added to incubation flasks containing male rat liver microsomes, and aniline, pentobarbital, and benzpyrene hydroxylase activity were measured. Microsomes from 300 and 100 mg liver for aniline and pentobarbital hydroxylase, respectively, were incubated with various substrates, with and without ethanol. It was shown that chronic ethanol

feeding in rats produced hepatic steosis (HS), proliferation of hepatic smooth endoplasmic reticulum (HSER), and increased microsomal activity, accompanied by increased hepatic aniline, pentobarbital, and benzpyrene hydroxylase activity. In humans, an ethanol diet produced HS, proliferated HSER, and a 2-3-fold increase of hepatic pentobarbital hydroxylase activity. Ethanol in vitro inhibited microsomal drug-metabolizing enzyme activity. These results may partially explain the clinical observation that sober alcoholics are more resistant to the action of drugs such as barbiturates, whereas intoxicated individuals are more sensitive.

1166. Rubin, E., Gang, H., Misra, P. S., and Lieber, C. S.

INHIBITION OF DRUG METABOLISM BY ACUTE ETHANOL INTOXICATION: A HEPATIC MICROSOMAL MECHANISM.

Amer. J. Med. (New York), 49: 801-806 (30 ref.),

1970.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – in vitro – blood lev. – absorp., distrib., stor. – liver, kidney – metab. proc. – barbiturates – enzymes – tranquilizers – *CAAAL-0 B-0979.

Activities of aniline, pentobarbital, and benzpyrene hydroxylases, aminopyrene and ethylmorphine demethylases, and NADPH-cytochrome C and cytochrome P₄₅₀ reductases were measured in hepatic microsomes from rat liver homogenates. All were inhibited by addition of ethanol to the medium, aniline hydroxylase being the most sensitive. Ethanol (10, 50, and 100 mM) also reduced the rate of metabolism of meprobamate (0.3 mM) by rat liver slices, apparently due to inhibition of the enzymes. This inhibition was competitive or partly competitive. ¹⁴C-pentobarbital (40 mg/kg) was injected ip into rats 2 hr after 5 g/kg ethanol by gastric intubation. Disappearance of pentobarbital from blood and total body was retarded. 4 non-alcoholic human volunteers were given 6 mg/kg ¹⁴C-pentobarbital po, followed 16 hr later by an initial dose of 1 g/kg 95% ethanol in fruit juice, and thereafter by doses of 24 g ethanol every 2 hr until 34-40 hr after pentobarbital administration. One week later, a similar experiment was performed, using 12-15 mg/kg meprobamate po; ethanol administration (1 g/kg initially, followed by 24 g every 2 hr) was begun after 14 hr, and extended to 24 hr post-meprobamate. It was found that the half-life of meprobamate was increased 2-5-fold. It is concluded that the mechanism of ethanol-induced inhibition of drug-metabolizing enzymes may be related to the interaction of ethanol and hepatic microsomes, as exemplified by microsomal ethanol oxidation and the binding of ethanol to microsomal hemoprotein.

1167. Rubin, E., Lieber, C. S., Alvares, A., Levin, W., and Kuntzman, R.

INTERACTION OF ETHANOL AND MICROSOMAL HEME PROTEIN: ITS EFFECT ON HUMAN DRUG METABOLISM.

Amer. J. Path. (New York), 59(3): 55a (0 ref.),

1970.

E – abst. – exp. cont. – cross-tol. – humans – mammals – chronic admin. – in vivo – in vitro – liver, kidney – metab. proc. – barbiturates – miscellaneous – *CAAAL-0 B-0980.

Human volunteers were given ethanol (42-48% of total cal) for 3-4 weeks. It was found that the plasma half-life of several drugs, including pentobarbital, was substantially decreased after ethanol feeding, and that the blood ethanol clearance rate was accelerated. In another experiment, volunteers were given pentobarbital, and plasma drug disappearance rate was determined. A large ethanol dose resulted in a considerably reduced rate of drug disappearance, probably through inhibition of hepatic pentobarbital hydroxylase. In a third experiment, ethanol was added to hepatic microsomes from ethanol-treated and pair-fed control rats. The addition of ethanol resulted in spectral changes, with a trough at 385 mμ, and a peak at 415 mμ. The magnitude of the ethanol peak was tripled in the ethanol group, while P450 was increased by 82%. The increase in spectral shift by chronic ethanol pretreatment indicates an in vivo effect, probably by the binding of ethanol to microsomal heme protein. Ethanol weakly inhibited the binding of aniline to P450, but had no effect on hexobarbital binding, although 70-100 mM ethanol inhibited P450 reductase by 30%.

1168. Rubin, E., Gang, H., and Lieber, C. S.
 INTERACTION OF ETHANOL AND PYRAZOLE WITH HEPATIC MICROSOMES.
 Biochem. Biophys. Res. Commun. (New York), 42(1): 1-8 (18 ref.), 1971.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – in vitro
 – absorp., distrib., stor. – liver, kidney – metab. proc. – barbiturates – *CAAAL-0 B-0981.

Hepatic microsomes were obtained from 150-200 g male Sprague-Dawley rats. The hemoglobin was removed, and the microsomes diluted to a concentration of 2.5 mg protein/ml. The difference spectrum produced by the binding of pyrazole to these microsomes, and to those obtained from rats fed ethanol for 24 days, was determined in a dual wave-length spectrophotometer. Ethanol was added to microsomal suspension, and the spectral change recorded. Pyrazole was added to 6 ml of the suspension; to 3 ml of this, ethanol was added, and the spectral change recorded. The effects of hexobarbital and aniline on the magnitude of spectral changes induced by ethanol binding were similarly studied. The rate of reduction of cytochrome P450 was determined. Prior exposure of microsomes to hexobarbital (a type 1 binder) had no effect on the spectral change, while pyrazole or aniline (type 2 binders) diminished it. The spectral change was doubled by ethanol consumption. It was found that pyrazole inhibits drug-metabolizing enzymes and binds to hemoprotein, resulting in a type 2 spectrum. Binding was unaffected by agents yielding a type 1 spectrum. Both ethanol and pyrazole inhibited reduction of cytochrome P450.

1169. Rudolf, W.
 UM DIE PROTEKTIVE ANGST. [On the protective fear].
 Med. Klin. (Munich), 52(25): 1120-1121 (3 ref.), 1957.
 G – general – review – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – mammals – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – CNS – sed., hypnot. – tranquilizers – *CAAAL-0 A-1078.

A few works on the effect of tranquilizers, alone and in combination with alcohol, on man and mice are cited and discussed. Tests on aircraft pilots are mentioned which showed that tranquilizers impair reaction time, coordination, judgement, memory, and in particular, suppress what is termed the "protective fear". In other tests on human subjects, the simultaneous intake of tranquilizers and alcohol has caused disinhibition, aggression, speech difficulties, and a real increase in blood alcohol concentration as compared with determinations made on the same subjects on a 2nd occasion (alcohol intake only).

1170. Rummel, W., and Schmitz, T.
 DIE ANTICURAREWIRKUNG DES ALKOHOLS. [The anticurare effect of alcohol].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 222: 257-261 (14 ref.), 1954.
 G – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – in vitro – dose resp. – G.I. tract – nerv. syst. – skel., muscle, skin – elect., water-bal. agents – musculoskel. agents – *CAAAL-7119-D2 A-1079.

The effects of various concentrations of alcohol on the electrical stimulation of the isolated rat phrenic-diaphragm preparation were studied. Lower alcohol concentrations (1.28% or 0.4 ml 96% alcohol in 30 ml) stimulated the endplate, whereas higher ones (2.56% or 0.8 ml) inhibited the effect; an intermediate dose (1.92% or 0.6 ml) caused a brief stimulation of a single contraction, but had no further effect. Inhibiting and stimulating alcohol concentrations antagonized curare inhibition, and paralyzing curare concentrations antagonized paralyzing alcohol concentrations. To see if the alcohol effect might be due to a depolarizing action, non-paralyzing concentrations of alcohol (1.92%) and potassium (which has a known depolarizing action) were applied in combination, and an additive paralyzing effect was noted. Even a stimulating alcohol concentration (52% potassium chloride)

resulted in a sudden lowering of contract amplitude. The fact that alcohol and curare act antagonistically suggests a different mechanism of action.

1171. Runeburg

KRONISK BLYFÖRGIFTNING, KOMPLICERAD MED KRONISK ALKOHOLISM.

[Chronic lead poisoning complicated by chronic alcoholism.].

Finska Läkaresällskapet, Handlingar (Helsinki), 22: 439-441 (O ref.),

1880.

S – SEC – abst. – general – case hist. – DC (unspec.) – drug-dep. humans – CNS – G.I. tract – nerv. syst. – skel., muscle, skin – *CAAAL-0

A-1315.

A case of lead poisoning complicated by chronic alcoholism in a 47 yr-old man is reported. The patient had been exposed to lead vapours in his work. Intermittent pains in the arms, weakness, abdominal pains, and constipation were the symptoms first noticed, and these were attributed to a bad cold. However, over a period of a few weeks, the pains persisted and worsened, and spread to involve his legs as well as his arms. Upon reporting to a clinic, a diagnosis of chronic lead poisoning complicated by alcohol was made. The patient at this time exhibited a grayish pallor of the skin, muscular atrophy, malnutrition, unsure movements, and slight tremor. A treatment program consisting of potassium iodide administration, warm baths, and electric therapy was initiated. The tremor and other symptoms gradually disappeared, but a considerable residual atrophy of the shoulder muscles left him unable to lift his arms to normal height. The author does not draw any connection between the alcoholism and lead poisoning, yet, it is noted that the patient had previously consumed spirits in large quantities but had reduced his intake in the last few years, and that the symptoms first began to appear 4 yr ago approximately at the same time as the reduction of alcohol intake.

1172. Runkevich, M.

VLIIANIE STRIKHNINA NA OTRAVLENIE ALKOGOLEM. [Effect of strychnine on alcoholic intoxication].

Tomsk Universitet, Izvestia (Tomsk), 9(7): 1-46 + 23 graphs (76 ref.),

1896.

R – exp. cont. – DC (decrease) – mammals – acute admin. – chronic admin. – in vivo – cardiovasc. – CNS – respir. – stimulants – *CAAAL-0

A-1080.

A series of controlled experiments was carried out on dogs to study the effect of strychnine on alcohol poisoning. The author concludes that strychnine increases the activity of psychomotor centers depressed by alcohol. It sharply intensifies heart activity, raises blood pressure during alcohol narcosis, and stimulates the respiration. Strychnine raises the body temperature which is decreased under the influence of alcohol, but only with doses large enough to cause convulsions. Although these conclusions apply only to animals poisoned by alcohol, the author feels that strychnine exercises a similar influence on chronic alcohol addiction—especially with respect to the heart, blood pressure, respiration, and activity of psychomotor centers. Strychnine is more effective on the chronic stage than on the acute stage of alcohol poisoning. Considerable tabulated and plotted experimental data are presented.

1173. Russek, H. I., Zohman, B. L., and Dorset, V. J.

EFFECTS OF TOBACCO AND WHISKEY ON THE CARDIOVASCULAR SYSTEM.

J.A.M.A. (Chicago), 157(7): 563-568 (12 ref.),

1955.

E – exp. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – cardiovasc. – *CAAAL-7102-D1

A-1081.

The response of the heart to nicotine was investigated in 2 groups of subjects: (1) 28 normal habitual smokers, aged 21-60, with unexplained precordial pain or with a history of symptoms from smoking, such as palpitations and nausea; and (2) 37 patients with coronary disease, aged 42-70, showing deterioration in the form of the ballisto-cardiogram after smoking. Whiskey was found to prevent

ballistic changes induced by smoking, probably due to the vasodilating action of the former on peripheral vessels. There is, however, no possibility that alcohol can vitiate the adverse effects of nicotine on the cardiovascular system. The protection afforded the heart by whiskey during smoking is purely an illusion. Whiskey is not a coronary vasodilator, nor is nicotine a coronary vasoconstrictor. The findings, therefore, are interpreted as confirming the clinical opinion that whiskey tends to antagonize tobacco only with respect to peripheral circulation.

1174. Rutenfranz, J., and Jansen, G.

ÜBER DIE KOMPENSATION VON ALKOHOLWIRKUNGEN DURCH COFFEIN UND PERVITIN BEI EINER PSYCHOMOTORISCHEN LEISTUNG. [On compensation of the alcohol effect by caffeine and pervitin in a psychomotor performance].

Int. Z. Angew. Physiol. (Berlin), 18: 62-81 (30 ref.), 1959.

G – exp. cont. – exp. comp. – DC (decrease) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – CNS – amphetamines – stimulants – *CAAAL-9479-J1 A-1082.

2 male human subjects were exposed to the following experimental conditions: when sober, after 0.5 or 1.0 g/kg alcohol po, and after 0.5 or 1.0 g/kg alcohol po plus an injection of 0.2 g caffeine or 9 mg pervitin. Performance was rated on an automobile driving device. The smaller alcohol dose almost immediately began to impair performance, reaching a peak of impairment 70 min later. Pervitin reversed the alcohol effect—perception was normal and driving was unimpaired; at a blood alcohol concentration of 0.6°/oo, the subjects felt no impairment. Caffeine reversed the alcohol effect to a lesser degree. After the larger alcohol dose, pervitin improved performance, but did not entirely counteract alcohol impairment. Neither drug affected the alcohol metabolism, and other functions, such as balance and self-criticism, remained impaired. It is concluded that the results of tests on certain functions do not warrant generalizations concerning other functions.

1175. Ryback, R. S.

EFFECT OF ETHANOL, BOURBON AND VARIOUS ETHANOL LEVELS ON Y-MAZE LEARNING IN THE GOLDFISH.

Psychopharmacologia (Berlin), 14: 305-314 (18 ref.), 1969.

E – exp. cont. – exp. comp. – congen. stud. – other org. – dose resp. – blood lev. – CNS – alcohols – antispasmodics – *CAAAL-0 B-0440.

2 experiments were performed to find out the effect of ethanol and of ethanol in combination with congeners on the learning ability of goldfish placed in test sol. The first experiment used synthetic ethanol sol (400 mg/100 ml and 650 mg/100 ml), which were compared with water controls, and the second test compared ethanol sol with a low congener content (400 mg/100 ml, 550 mg/100 ml, and 650 ml/100 ml) to a bourbon sol of 650 mg/100 ml with a high congener content. It is concluded that there is a difference between the behavioural effect of ethanol (with a lower congener content) and bourbon (with a high congener content). Fish in the bourbon sol were poorer learners than fish in ethanol sol. It is possible that congener alcohols potentiate the depressive effects of higher doses of ethanol, but this is not yet established.

1176. Ryback, R. S., and Dowd, P. J.

AFTEREFFECTS OF VARIOUS ALCOHOLIC BEVERAGES ON POSITIONAL NYSTAGMUS AND CORIOLIS ACCELERATION.

Aerospace Med. (St. Paul), 41(4) : 429-435 (38 ref.), 1970.

E – exp. comp. – congen. stud. – mot. vehic. – humans – acute admin. – in vivo – mot. perform. – CNS – nerv. syst. – *CAAAL-0 B-0555.

The after-effects of bourbon and vodka on subjective tumbling and positional and coriolis nystagmus were tested. Reactions were recorded by electronystagmography. 3 groups were tested: pilots, non-

flying personnel, and non-flying personnel accustomed to coriolis stimulation, and their baseline values were determined. On the evening of the tests, each subject consumed either .85 or 1.7 ml ethanol/kg, the high dose being given to those who usually consumed more than 20 oz of hard liquor per week. 10 1/2 hr later (the next morning), the subjects were re-tested. When values deviated from the control levels, tests were again performed the following morning. In analyzing the results, the researchers looked for the extent of positional alcohol nystagmus I and II (PAN I and PAN II). It was found that PAN II was present 34 hr after both high and low doses of bourbon, but only after high doses of vodka. In one subject, PAN I was present 10 hr after vodka. Among subjects tested on both vodka and bourbon separately, PAN II was found to last longer and ocular overshoot was more consistent after bourbon. There was also increased vestibulo-ocular response to coriolis acceleration 38 hr after bourbon ingestion, an effect which was absent in the post-vodka group.

1177. Ryback, R. S., and Ingle, D.

EFFECT OF ETHANOL AND BOURBON ON Y-MAZE LEARNING AND SHOCK AVOIDANCE IN THE GOLDFISH.

Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 136-141 (16 ref.), 1970.
E – exp. cont. – exp. comp. – congen. stud. – other org. – acute admin. – in vivo – blood lev. – psychol. perform. – CNS – *CAAAL-12895 B-0554.

Ethanol and bourbon were used to study the role of congeners in the behavioral response of goldfish to alcoholic beverages. 5 groups of 6 fish each were trained in a Y-maze until a criterion of 18 out of 20 correct turns was obtained. 1 group trained in water, and the remainder trained in a bourbon or ethanol sol of 650 mg/100 ml, after being in the same sol for 2 hr (2 groups) or 6 hr (2 groups). After 2 hr in bourbon or ethanol, the fish demonstrated a similar rate of learning. After 6 hr of exposure, the fish in the ethanol group performed similarly to the controls in water, whereas 5 of 6 fish in the bourbon group did not learn even after 2 consecutive days of training. In a second experiment, 16 goldfish were trained in a shock-avoidance procedure, and then rested after spending 3 hr in a 600 mg/100 ml sol of either ethanol or bourbon. The fish exposed to ethanol performed better than those exposed to bourbon. It is concluded that, after 3 and 6 hr of exposure to an alcoholic beverage, the congeners express a disruptive effect on learning behavior.

1178. Rydberg, S.

INHIBITION OF ETHANOL METABOLISM IN VITRO BY 4-iodo-PYRAZOLE.

Biochem. Pharmacol. (New York), 18: 2424-2428 (8 ref.), 1969.
E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – metab. proc. – *CAAAL-0 B-0982.

The in vivo effect of 4-iodopyrazole on ethanol metabolism was studied in rats. 32 female rats (200-235 g) were given 32.6 m moles/kg ethanol in 12% (w/v) sol ip, alone or in combination with various doses of 4-iodopyrazole or pyrazole. Blood samples were taken at 30 min, and then at 60 min, intervals, and were analyzed for ethanol by the Widmark and gas chromatographic methods for 5-28 hr. The degree of inhibition of the rate of ethanol metabolism by 4-iodopyrazole varied between 19 and 84%, and the ED₅₀ was found to be 0.27 m moles/kg. The presence of ethanol in blood was prolonged from 4 hr in controls to 28 hr after 1.25 m moles/kg 4-iodopyrazole. The ED₅₀ of pyrazole was of the same order of magnitude, or 0.43 m moles/kg. It is conjectured that the discrepancy in activity between 4-iodopyrazole in vivo and in vitro may be due to the difference in pH of the solutions of pyrazole and 4-iodopyrazole administered, or to differences in absorption and distribution within the organism.

1179. Saar, H., and Paulus, W.

UNTERSUCHUNGEN ÜBER DIE REDUZIERENDE WIRKUNG EINIGER DESINFEKTIONSMITTEL. (EIN BEITRAG ZUR HAUTDESINFEKTION FÜR DIE

BLUTENTNAHME ZUR ALKOHOLBESTIMMUNG.) [Investigations of the reducing effect of some disinfectants. (A contribution on skin disinfection for blood extraction for the purpose of alcohol determination)].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 34: 467-470 (0 ref.), 1941.
G – exp. comp. – humans – acute admin. – in vivo – blood lev. – anesthetics – *CAAAL-0
A-1083.

It was questioned whether disinfection of the skin with alcohol or other disinfectants containing alcohol, such as tincture of iodine, soap spirit, ether, benzine, benzol, zephirol, sagrotan, lysoform, or lysol, could influence the blood alcohol level when the sample was taken by venula. Experiments with sober subjects under the most unfavourable conditions (application of disinfectants in far higher amounts than usual) showed that the blood alcohol level caused by contamination of the blood by disinfectants stayed far below 0.1°/oo.

1180. Sachdev, K. S., Panjwani, M. H., and Joseph, A. D.
POTENTIATION OF THE RESPONSE TO ACETYLCHOLINE ON THE FROG'S RECTUS ABDOMINIS BY ETHYL ALCOHOL.

Arch. Int. Pharmacodyn. (Gand), 145(1-2): 36-43 (4 ref.), 1963.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – other org. – in vitro – dose resp. – nerv. syst. – skel., muscle, skin – autonomic agents – cardiovasc. agents – diagnost. agents – *CAAAL-0
A-1436.

Studied were the actions of ethanol on the following: the contraction produced by acetylcholine and carbachol on the frog rectus abdominis, responses of isolated frog sciatic gastrocnemius preparation to electric current, the contraction produced by acetylcholine and histamine on guinea pig ileum, and adrenalin-induced guinea pig seminal vesicle contractions. Various concentrations of ethanol markedly potentiated responses to acetylcholine (0.2-2 mcg/cc) and carbachol (0.5-3 mcg/cc) by frog rectus abdominis. The actions of concentrations of ethanol varying between .005 and .02 M on acetylcholine and histamine responses of guinea pig ileum were similar—.005-.01 M did not affect the height of contraction, while .01-.02 depressed the contractions. None of the ethanol concentrations (.02-.5 M) significantly depressed or potentiated the adrenaline response of the guinea pig seminal vesicle. It is suggested that the marked potentiation by ethanol of acetylcholine response on striated muscle, and of acetylcholine and histamine response on smooth muscle, may be due to irritation preceeding the protein precipitant action.

1181. Sachdev, K. S., Rana, P. K., Dave, K. C., and Joseph, A. D.
A STUDY OF THE MECHANISM OF ACTION OF THE POTENTIATION BY ALIPHATIC ALCOHOLS OF THE ACETYLCHOLINE RESPONSE ON THE FROG'S RECTUS ABDOMINIS.

Arch. Int. Pharmacodyn. (Gand), 152(3-4): 408-415 (6 ref.), 1964.
E – exp. comp. – DC (add., infra-add., unspec. incr.) – other org. – acute admin. – in vitro – dose resp. – nerv. syst. – skel., muscle, skin – autonomic agents – *CAAAL-0
A-1437.

Frog rectus abdominis was bathed in vitro, and the effects on its response to acetylcholine (Ach) were tested by addition of: aliphatic alcohols, potassium and calcium hydroxide, potassium and calcium chloride, ferrous sulphate, and tannic acid, at various concentrations. Methyl, ethyl, n-propyl, isopropyl, butyl, and amyl alcohols potentiated the response to Ach, as did the hydroxides. Ferrous sulphate and tannic acid (protein precipitants) had no such action. The effects of aliphatic alcohols on frog sciatic gastrocnemius twitch responses to electric stimulation were tested, and were found to have varied effects. Thresholds of irritation were established by finding the concentration at which alcohols, when applied to the lower limb of a spinal frog, produced a reflex withdrawal in a specified time. The concentrations of minimal potentiation were 1/100, 1/40, 1/150, 1/100, 1/20, and 1/20 of the threshold concentration for irritation, for ethyl, methyl, propyl, isopropyl, butyl, and amyl alcohols,

respectively. It is suggested that the potentiating action of aliphatic alcohols on Ach may be due to the OH⁻ group. The concentrations of alcohols causing irritation and precipitation of proteins are much higher than those producing potentiation. This phenomenon is probably not due to irritation.

1182. Saidkasymov, T.

ANTAGONIZM ERVININA K ETILOVOMU ALKOGOLIU. [Antagonism of ervinin to ethyl alcohol].

Meditinskii Zhurnal Uzbekistana (Tashkent), 3: 56-59 (4 ref.), 1963.
R – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – species or sex diff. – CNS – stimulants – *CAAAL-10775-D2
A-1084.

54 rabbits received 4 or 5 ml 96% alcohol/kg in a 25% sol po, plus 5-20 mg/kg ervinin iv. In 15 controls receiving alcohol only, the 4 ml alcohol dose caused narcosis of 40-90 min, and the 5 ml dose caused a 90-240 min narcosis. 8 mg/kg ervinin brought about a brief awakening, 10-15 mg/kg doses caused the animals to sit up for prolonged periods, and 20 mg/kg abolished narcosis and caused some seizures. 5-20 mg/kg corazole was also given after alcohol; 15 mg/kg shortened alcohol narcosis by 1 hr, but the animals later relapsed into coma which lasted as long as in the controls. 4 ml 96% alcohol/kg ip caused a narcosis of 8-20 min in mice. 50-300 mg/kg ervinin ip given simultaneously had the following effects: 50 mg/kg slightly shortened narcosis, 100 mg/kg prevented narcosis in 60% of the mice, doses of up to 150 mg/kg prevented it in 80%, and doses greater than 150 mg/kg appeared to enhance, rather than antagonize the alcohol effect.

1183. Salant, W.

THE COMPARATIVE TOXICITY OF ETHYL AND AMYL ALCOHOL AND THEIR EFFECT ON BLOOD PRESSURE.

Proc. Soc. Exp. Biol. Med. (New York), 6: 134-135 (O ref.), 1908-09.
E – SEC – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – other org. – acute admin. – in vivo – cardiovasc. – CNS – stimulants – *CAAAL-0
A-1351.

The comparative toxicities of ethyl (E) and amyl (A) alcohols were studied in frogs, rabbits, cats, and dogs, and the effects of E and A on blood pressure were compared in dogs and cats. It was found that the toxicity of A was in all cases much greater than that of E, and the fall in blood pressure after A was considerably more than that induced by E. Recovery from the effects of A was also slower than from E. Some observations on the effects of caffeine on the depressant action of A and E were made. The injection of 25-50 cc 2% caffeine sol after iv administration of 2% E or A sol was found to retard recovery of blood pressure in both cases by 15-20 min.

1184. Saldeen, T., and Johansson, Ö.

THE SIGNIFICANCE OF CHRONIC HEART DISEASE, FATTY LIVER, AND CONSUMPTION OF BARBITURATE AND LIBRIUM ON THE TOLERANCE TO ETHYL ALCOHOL, AS JUDGED IN A POSTMORTEM SERIES.

J. Forensic Sci. (Mundelein), 12(3): 273-294 (45 ref.), 1967.
E – stat. surv. – DC (add., infra-add., unspec. incr.) – med.-leg. – post-mort. – humans – drug-dep. humans – blood lev. – other drug lev. – cardiovasc. – CNS – liver, kidney – respir. – barbiturates – tranquilizers – *CAAAL-0
B-0441.

Of 5,100 autopsies between 1957 and 1965, 82 cases were found to have a blood alcohol concentration of 0.04%, and 52 cases showed the presence of both alcohol and barbituric acid derivatives. In the alcohol-barbiturate poisonings, the blood alcohol concentrations were substantially lower than in poisonings from alcohol alone; the blood concentrations of both substances were low in some cases, especially with amobarbital-alcohol, whereas, with pentobarbital-alcohol and phenobarbital-alcohol,

concentrations of both substances were high—in 3 cases, the pentobarbital concentrations in the liver were 12.0, 17.4, and 20 mg/100 g, and the blood alcohol concentrations were 0.09, 0.16, and 0.18%. Death from combined intoxications occurred rapidly, and much more rapidly than would be expected from either substance alone. A few sudden deaths from consumption of meprobamate plus alcohol and heminevrin plus alcohol were also noted. In 4 cases, death occurred from combined intake of librium and alcohol.

1185. Salén, E. B.

OM GENGASEPOKEN I SVERIGE 1939-1945: EN KLINISK OCH SOCIAL-MEDICINSK ÖVERSIKT. [On the gengas period in Sweden 1939-1945: a clinical and socio-medical study]. Nord. Med. (Stockholm), 30(17): 923-931 and 933-934 (35 ref.), 1946.
S – ES – general – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – CNS – indust. intox. – *CAAAL-0 A-1085.

During World War II, due to an acute gasoline shortage, Sweden switched to the use of carbon monoxide on a large scale; by 1942 the number of CO-fuel or “gengas” (generator gas) automobiles had reached 100,000. The author begins with a historical and statistical analysis of generator gas poisoning in Sweden, including definition, diagnosis, and symptomatology. He points out that a symptom of CO-intoxication is an appreciably reduced tolerance to alcohol; this was observed under clinical conditions, and in a series of tests in which blood alcohol curves were measured under controlled conditions. The results indicated abnormally high blood alcohol values in the CO-intoxicated group, compared to the control. These high levels prolonged and appreciably strengthened the pathological effect of intoxication in 20% of the 150 persons tested, and more moderately affected others. The mechanism of this reaction is not elucidated, but the author contends that central nervous lesions are involved. He advocates testing the CO-Hb blood level, in addition to the blood alcohol test, in impaired driving accidents.

1186. Samochowiec, L., Wójcicki, J., and Dominiczak, K.

DER EINFLUSS DER „ESSENTIELLEN“ PHOSPHOLIPIDE AUF LEBERVERÄNDERUNGEN NACH CHRONISCHER ÄTHYLALKOHOL UND ALLYLISOSULFOCYANAT-INTOXIKATION BEI WEISSEN RATTEN. [The influence of “essential” phospholipids on hepatic lesions in white rats caused by chronic intoxication with ethanol and allyl isosulfocyanate]. Arzneimittelforschung (Aulendorf), 17(11): 1374-1376 (18 ref.), 1967.
G – ES – exp. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. – in vivo – blood comp., sites, lymph – liver, kidney – anti-infectants – *CAAAL-0 B-0442.

In 60 white rats the influence of “essential” phospholipids (EPL) on the changes caused by chronic administration of ethanol (20% ethanol sol, 1.5 ml/100 g po) and allyl isosulfocyanate (AIS) (0.002 mg/100 g po) was studied. The serum controls revealed an increase of the esterified fatty acids and cholesterol (increase between 254 and 417%), and a highly positive turbidity test; histologically, a fatty degeneration of the liver was proved, which was most marked after combined administration of alcohol and AIS. That EPL can prevent such changes was proved by biochemical studies of the serum and histological examination of the liver.

1187. Sand

RECHERCHES EXPÉRIMENTALES SUR L'INTOXICATION PAR L'ALCOOL-ÉTHÉR. [Experimental investigation of intoxication by alcohol-ether]. Presse Médicale Belge (Brussels), 65(19): 440-444 (3 ref.), 1913.
F – exp. – DC (unspec.) – mammals – chronic admin. – in vivo – cardiovasc. – CNS – liver, kidney – respir. – anesthetics – *CAAAL-0 A-1086.

A series of experimental investigations were undertaken to determine the effect of alcohol-ether vapours on the animal organism. The experimental animals (dogs) were left at liberty during the day, but locked up from 8 o'clock in the evening until 6 o'clock in the morning in a room where 500 g of alcohol-ether contained in a flask were allowed to evaporate each night. After 15 days of intoxication, the animals died. An autopsy revealed basilar meninx hemorrhage, capillary hemorrhage, and a fatty degeneration of the biliary tract epithelium. The author concludes that, after the initial intolerance (excitation, attacks), an apparent tolerance is developed for alcohol-ether which masks progressively more serious lesions of the various viscera (pulmonary sclerosis, cirrhosis of the liver, nephritis).

1188. Sandberg, F.

A QUANTITATIVE STUDY ON THE ALCOHOL-BARBITURATE SYNERGISM.

Acta Physiol. Scand. (Stockholm), 22: 311-325 (16 ref.),

1951.

E – exp. cont. – exp. comp. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – dose resp.
 – CNS – barbiturates – sed., hypnot. – *CAAAL-5783-D2 A-1087.

3 groups of 10 mice each were exposed to the following test conditions: 0.228 mg alcohol/l of air by inhalation in a bottle; 0.001 mmoles/kg sc of kemithal, hexobarbital, cyclobarbitol, amobarbital, thiopental, or 1-carbethoxymethyl-5,5-diallylbarbituric acid, followed by alcohol inhalation; or barbiturate injection only. For each barbiturate, a coefficient of synergism, $k(1/\log \text{ time (alcohol + barbiturate)})$, divided by $1/\log \text{ time of alcohol} + 1/\log \text{ time of barbiturate}$, was calculated for stages A, B, and C of anesthesia; thus, the greater the value of the coefficient, the higher the degree of synergism, and a coefficient of 0.5 or less indicated no synergism. The k values for stage A of anesthesia were: kemithal, 0.810; hexobarbital, 0.897; cyclobarbitol, 0.607; amobarbital, 0.876; thiopental, 0.676; and 1-carbethoxymethyl-5,5-diallylbarbituric acid, 0.832. The average percentages of induction times for stages A, B, and C of anesthesia after alcohol plus barbiturate, compared to alcohol alone, were: kemithal, 50.0%; hexobarbital, 44.0%; cyclobarbitol, 80.5%; amobarbital, 45.7%; thiopental, 58.5%; and 1-carbethoxymethyl-5,5-diallylbarbituric acid, 48.0%. All barbiturates, therefore, enhanced the alcohol effect to various (significant) degrees. It was then determined whether the synergistic effect of hexobarbital and cyclobarbitol was additive or potentiative; for an alcohol concentration of 0.107 mg/l and a barbiturate dose of 0.080 and 0.050 mmoles/kg, the synergism was found to be potentiative.

1189. Sandler, R., Vinnick, L., and Freinkel, N.

INTERRELATIONSHIPS BETWEEN ETHANOL AND PHENYLETHYLBIGUANIDE ON HEPATIC GLUCONEOGENESIS *IN VITRO*.

Life Sci. (Oxford), 7(2): 459-465 (19 ref.),

1968.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vitro – liver, kidney – metab. proc. – *CAAAL-0 B-0556.

The actions of ethanol and phenylethylbiguanide (PEBG) on hepatic gluconeogenesis were studied in vitro with rat liver slices. The liver slices, in sol containing 1 mM alanine-U-C¹⁴ (1 μ curie) and: (1) nothing else (control), (2) 10mM ethanol, (3) 0.8 mM PEBG, or (4) 10 mM ethanol plus 0.8 mM PEBG, were incubated for 90 min in a Dubnoff metabolic incubator or in a Warburg respirometer, so that determinations of alanine disposition and respiratory metabolism, respectively, could be made. It was found that when ethanol and PEBG were combined, an additive depression was exerted on oxygen consumption, uptake of alanine, release of glucose-C¹⁴, and evolution of C¹⁴O₂. The effect of ethanol predominated on the production of lactic acid-C¹⁴ (increased) and aspartic acid-C¹⁴ (decreased), while the effect of PEBG predominated on glyceride-glycerol-C¹⁴ production (decreased). It is concluded that the inhibition of hepatic gluconeogenesis by ethanol and phenylethylbiguanide may involve different mechanisms, and that their combination can exert an additive depression on gluconeogenesis.

1190. Sankar, D. V. S., Gold, E., Sankar, B., and McRorie, N.
 EFFECT OF PSYCHOPHARMACOLOGICAL AGENTS ON DPN-DEPENDENT
 ENZYMES.
 Fed. Proc. (Bethesda), 20: 394 (0 ref.), 1961.
 E – abst. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute
 admin. – in vitro – liver, kidney – metab. proc. – antidepressants – hallucinogens – stimulants –
 *CAAAL-0 A-1438.

The effects of LSD, BOL, tyramine, phenylisopropyl hydrazide (JB516), 5-hydroxytryptophan (HTP), tryptamine, noradrenalin, d- and l-amphetamine, marsalid, and other compounds on enzymes requiring DPN as a coenzyme were investigated by following the increase in optical density at 340 mμ in the presence of the proper substrates. A maximum of 10⁻²M of the compounds was employed. Only LSD and BOL inhibited glutamic dehydrogenase, while JB516 showed a slight activation. HTP, tyramine, and tryptamine were found to inhibit yeast alcohol dehydrogenase; marsalid, the amphetamines, and a freshly-prepared JB516 sol were not inhibitory, although a JB516 sol allowed to stand for 4 hr showed marked inhibition. With vitamin A alcohol or ethanol as substrate, even a fresh JB516 sol was inhibitory; l-amphetamine exhibited much more inhibition than did d-amphetamine. JB516 inhibition was reversed by larger amounts of enzyme, DPN, or cysteine, showing that the inhibition is probably mediated through blocking of the sulphhydryl groups by JB516.

1191. Santesson, C. G.
 ÜBER DIE WIRKUNG VON ALKOHOL UND EINIGEN ANDEREN GIFTEN AUF DIE
 HERZHEMMUNG BEIM FROSCH. [The effect of alcohol and some other poisons on cardiac
 inhibition in the frog].
 Scandinavisches Archiv für Physiologie (Berlin), 69: 255-292 (55 ref.), 1934.
 G – SEC – exp. cont. – DC (unchanged) – other org. – acute admin. – in vitro – dose resp. – cardiovasc.
 – stimulants – tranquilizers – *CAAAL-0 A-1457.

The effect of certain drugs on muscarine- or acetylcholine-induced inhibition of heart functions was investigated in frogs. Several drops of muscarine platinous chloride (0.08-0.1% sol) or acetylcholine (1% sol) were applied directly to the heart of *Rana temporaria* in situ. Then drops of solutions of various test drugs, including alcohol (0.2, 1.0, 2.5, 5, and 10% sol) were applied. Finally, the hearts were rinsed with Ringer's sol, and atropine was given to relieve the inhibition. The results were tabulated and graphically expressed. Neither low nor high alcohol concentrations affected the inhibiting action of muscarine or acetylcholine. From the results, and from the findings of other researchers, it is concluded that there is little possibility that alcohol can effectively antagonize drug-induced inhibition of any body functions. Nevertheless, it is pointed out that, since the stimulating effect of small doses of alcohol is known, it would be erroneous to conclude definitely that alcohol is a true paralyzing substance.

1192. Sautet, J., Desanti, E., Payan, H., Aldighieri, J., Gay, R., Arnaud, G., Deluy, M., Castelli, P., and Rampal, C.
 CONTRIBUTION À L'ÉTUDE EXPÉRIMENTALE DE L'ACTION DE DIVERS VINS ET
 ALCOOLS CHEZ LA SOURIS BLANCHE (POIDS, MORTALITÉ, MÉTABOLISME
 BASAL, CONTRACTION MUSCULAIRE, ANATOMO-PATHOLOGIE). [Contribution to
 the experimental study of the action of various wines and alcohols in the white mouse (weight,
 mortality, basal metabolism, muscular contraction, anatomo-pathology)].
 Revue de Pathologie Générale et de Physiologie Clinique (Paris), 60: 1623-1673 (14 ref.), 1960.
 F – exp. cont. – exp. comp. – congen. stud. – mammals – acute admin. – chronic admin. – in vivo
 – CNS – metab. proc. – skel., muscle, skin – *CAAAL-10053-D2 A-1439.

Experiments concerning effects of wines on mortality, weight gain, basal metabolism, and muscular fatigue, conducted by the authors since 1955, are reviewed. In 1 experiment, 400 male and female

white mice (20 g) received a daily regimen of bread and water, or bread and water plus: 1 cc 10% alcohol sol (corresponding to 40 g/kg 10% wine), 1 cc 10% red wine (corresponding to 40 g/kg), or 1 cc dealcoholized wine. It was found that the control rats showed a regular and progressive wt gain, whereas the other groups showed an irregular course of wt gain. Mortality was 0% in controls, 15.4% in the alcohol group, 11.5% in the wine group, and 4.6% in the dealcoholized wine group. The results of further experiments showed that, of sweet wine, diluted sweet wine, diluted sweet wine with sodium glycolate, or water with glycolate, sweet wine caused the greatest mortality, and produced the most muscle fatigue. Wt gain was highest in water-fed animals.

1193. Sbertoli, C., and Brambilla, G.

TRE CASI DI INTOLLERANZA ALL'ALCOOL COME UNICO SINTOMO DELLA INTOSSICAZIONE DA TRICLOROETILENE. [Three cases of alcohol intolerance as the only symptom of trichloroethylene intoxication].

Med. Lavoro (Milan), 53(5): 353-358 (13 ref.),

1962.

I - ES - GS - general - case hist. - DC (sensit.) - humans - cardiovasc. - CNS - metab. proc. - senses - analg., antipyret. - anesthetics - *CAAAL-0 A-1440.

3 cases of industrial trichloroethylene (TCE) poisoning, resulting in intolerance to alcohol, are described. In 2 of the cases, the reaction to alcohol resulted from exposure to TCE for 2 weeks in 1 case, and for 2 days in the other. In the third case, acute TCE poisoning developed after exposure to the substance for a few months, in addition to exposure to tetramethylthiuram disulphide (TMTD). The first 2 men were moderate users of alcohol, while the third was a heavy drinker. The TCE symptoms were similar to the antabuse-alcohol reaction—a red flushing of the face and neck, nausea, a heat sensation, etc; the effects lasted for about 1 hr after exposure, following which no reaction to alcohol was experienced. The TMTD symptoms were: strong pains, trembling, a sensation of constriction in the throat, a feeling of heat in the face, erythema, tachycardia, and a temporary increase in arterial pressure. These effects also lasted for about 1 hr, but they persistently reappeared for 3 months after exposure when alcohol was ingested. The author states that TCE is preferentially metabolized by acetaldehyde dehydrogenase (ADH), resulting in an acetaldehyde accumulation, whereas TMTD directly inhibits ADH, resulting in an acetaldehyde accumulation as well, but with more persistent effects.

1194. Scarlato, G., and Menozzi, C.

AZIONE DEL MEPROBAMATO SULLA INTOSSICAZIONE CRONICA SPERIMENTALE DA ALCOOL: STUDIO ISTOLOGICO ED ISTOCIMICO DEL SISTEMA NERVOSO.

[Effect of meprobamate on experimental chronic alcohol intoxication: histological and histochemical study of the nervous system].

Sist. Nerv. (Milan), 11: 165-174 (21 ref.),

1959.

I - ES - exp. cont. - DC (decrease) - DC (unchanged) - mammals - chronic admin. - in vivo - CNS - nerv. syst. - tranquilizers - *CAAAL-9098-G2 A-1088.

24 rabbits were given 1 of the following treatments: (1) iv injection of 1.5 cc 95% alcohol for 19 days, (2) the above plus im meprobamate (120 mg/kg) beginning on the 20th day and continuing for 19 days thereafter, and (3) the above drugs simultaneously for 19 days in the above dosages. There was no substantial clinical or histological difference between rabbits of groups (1) and (2); rabbits in group (3), however, showed fewer clinical signs than the other groups (no diarrhoea, and body weight within normal limits). Simultaneous administration of meprobamate afforded some protection against the neural and glial alterations of nervous tissue arising from chronic intoxication—microglial proliferation was absent in nerve cells, and the ribonucleic acid content was normal, and in some cases slightly increased. Meprobamate did not afford protection against the ataxia caused by alcohol.

1195. Schelb, H.

EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE WIRKUNG PROTRAHIERTER ALKOHOLAUFNAHME AUF DIE LEISTUNGSFÄHIGKEIT UND ÜBER DIE

„ERNÜCHTERNDE“ WIRKUNG VON KAFFEE. [Experimental investigations on the effect of protracted alcohol ingestion on performance, and on the “sobering-up” effect of coffee].

Dissertation, Medical Faculty of the University of Göttingen, Germany, 55 pp. (53 ref.), 1939.

G – exp. cont. – DC (decrease) – humans – chronic admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – CNS – stimulants – *CAAAL-0 A-1089.

The Giese reaction time test apparatus was used to determine (1) the influence of 117 g of alcohol po on attention, coordination, and reaction time in 2 human subjects, and (2) the influence of 0.45 g of caffeine po on the above alcohol effect. Results showed a definite performance impairment which was most pronounced when the blood alcohol concentration was highest. Caffeine reduced the psychic alcohol effects, but had no influence on the blood alcohol concentration itself. For 6 weeks, the course of the physiological poisoning remained unchanged in 1 subject; in the second subject, only a slight tolerance to alcohol and caffeine developed.

1196. Schiefgen, W.

BEOBSACHTUNGEN BEI KOMBINATIONSGABEN VON ALKOHOL UND INSIDON.

[Observations after administration of alcohol and insidon in combination].

Dissertation, Medical Faculty of the University of Bonn, West Germany, 33 pp. (6 ref.), 1963.

G – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – CNS – antidepressants – *CAAAL-0 A-1090.

7 human subjects (6 healthy and 1 suffering from paroxysmal stenocardia) were tested for reaction time and concentration. On the first day, they received no alcohol or insidon. On the second and fourth days, they received alcohol up to a blood alcohol level of between 0.58 and 1.18°/oo. On the third and fourth day, they received 200 mg insidon, which, on the fourth day, improved the performance of the subjects under the influence of alcohol. The author points out that these antagonistic effects were established for comparatively low alcohol levels and for a 20 min testing time. It is conceivable that insidon may even have an additive effect with higher blood alcohol levels.

1197. Schleyer, F.

ZUR ÄRZTLICHEN BEGUTACHTUNG VON TRUNKENHEITSDELIKTEN IN FORO.

[Expert court testimony by the physician in offenses involving drunkenness].

Öffentliche Gesundheitsdienst (Stuttgart), 12: 461-470 (13 ref.),

1951.

G – SEC – general – DC (unchanged) – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – blood lev. – mot. perform. – hallucinogens – stimulants – *CAAAL-5843-A15 A-1091.

The meaning of blood alcohol levels for physicians who may have to testify in court in impaired driving cases is discussed. The judicial, rather than the physiological, viewpoint is presented. Alcohol absorption, oxidation, and tolerance, brain trauma, and other variables are also discussed. Since it is generally accepted that an alcohol blood concentration of 1°/oo or more causes impairment, neither the expert nor the counsel for the defense should argue about such points as whether the established concentration was 2.1°/oo or 1.9°/oo. Narcosis by inhalation of ether can simulate blood alcohol levels up to 0.4°/oo. This has to be taken into consideration when the blood sample is taken after emergency surgery is performed. Caffeine may clear up the sensorium but cannot improve motor performance. In all fatal accidents, blood samples and urine samples should be taken; the blood samples will be valid if taken up to 48 hr after death.

1198. Schleyer, F., and Mallach, H. J.

EINATMUNG VON BRENNSPIRITUSDÄMPFEN UND BLUTALKOHOLGEHALT, VERSUCHSERGEBNISSE. [Inhalation of fuel alcohol fumes and blood alcohol level.

Experimental results].

Zbl. Arbeitsmed. (Darmstadt), 6(25): 90-91 (9 ref.),

1956.

G – exp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – CNS – alcohols – *CAAAL-7643-U1

A-1092.

2 humans were subjected to vapours of fuel alcohol (25 l of denatured fuel alcohol with 2.5% methanol and pyridine bases) in a closed room (66 cubic meters). Blood alcohol values remained negative for 2 hr when the subjects were given 25 cc of 33% brandy on an empty stomach. After further ingestion of 125 cc wine (13.3% alcohol content), blood alcohol levels corresponded to theoretically expected values (between 0.7 and 0.87‰), using Widmark's method for analysis. It was felt that the fuel alcohol produced a reduced tolerance to alcohol, i.e., the behaviour of the subjects corresponded to a higher blood alcohol level.

1199. Schleyer, F., and Janitzki, U.

VERSUCHE ÜBER DIE WIRKUNG DES MEGAPHENS AUF DEN

BLUTALKOHOLSPIEGEL. [Studies on the effect of megaphen on the blood alcohol level].

Arch. Int. Pharmacodyn. (Gand), 141(1-2): 254-261 (7 ref.),

1963.

G – ES – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – species or sex diff. – metab. proc. – tranquilizers – *CAAAL-10587-A2

A-1093.

Rabbits received 1.0 or 1.5 g/kg 30% alcohol sol, followed 30 min later by 3.6-5.0 mg/kg chlorpromazine iv. 15-100 min after chlorpromazine, the blood alcohol curve showed peaks and plateaus, and its total decrease was generally slower. Rats received 2 g/kg 30% alcohol sol im or ip, followed 50 min later by 7.5 mg/kg chlorpromazine. Other rats received 10 mg/kg chlorpromazine/day for 3 days, and, 24 hr after the last drug dose, were given 2 g/kg alcohol. Again, the course of the alcohol elimination was retarded by chlorpromazine, and the blood alcohol curves were abnormal with repeated peaks and plateaus. It is considered that the chlorpromazine effect is due to its sympatholytic properties. The medico-legal implications are discussed.

1200. Schlierf, G., Gunning, B., Uzawa, H., and Kinsell, L. W.

THE EFFECTS OF CALORICALLY EQUIVALENT AMOUNTS OF ETHANOL AND DRY WINE ON PLASMA LIPIDS, KETONES AND BLOOD SUGAR IN DIABETIC AND NONDIABETIC SUBJECTS.

Amer. J. Clin. Nutr. (Bethesda), 15: 85-89 (6 ref.),

1964.

E – exp. cont. – congen. stud. – humans – acute admin. – in vivo – blood lev. – blood comp., sites, lymph – metab. proc. – *CAAAL-0

A-1441.

5 non-insulin-dependent diabetics, 1 patient with a colloid goiter and arteriosclerosis, and 1 with moderate obesity, were studied in a metabolic ward. In order to evaluate the hyper- or hypoketonemic effects of alcohol, the fat: carbohydrate ratio in the diet was such as to produce some elevation of ketone levels. Fat was in saturated (coconut oil) or unsaturated (safflower oil) form. Adequate protein was included. Alcohol (usually 61.6 g/day) was given as an aqueous ethanol sol or as a calorically-equivalent amount of very dry wine at 1 or 2 hr intervals from 9 am to 9 pm. Plasma ketones, cholesterol, phospholipids, free fatty acids, glycerides, and blood sugar were measured. Ingestion of ethanol sol caused an elevation, transient in some persons, of blood ketone values. Wine (total carbohydrate content equal to 2.2 g/day) had a lesser hyperketonemic effect. Both drinks caused a rise in plasma cholesterol in some subjects, but the substitution of unsaturated for saturated fat protected against the hyperlipidemic effects of alcohol. The data confirm the expectation that, as most of the ethanol is metabolized to acetaldehyde, it is a potential ketone precursor.

1201. Schlungs, H.

DIE WIRKUNG VON ASPIRIN UND PYRAMIDON AUF DIE ALKOHOLKONZENTRATION DES BLUTES. [The effect of aspirin and pyramidon on the alcohol concentration of the blood].

Dissertation, Medical Academy of Düsseldorf, Germany, 23 pp. (26 ref.), 1938.
 G – exp. comp. – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – blood lev. – other drug lev. – psychol. perform. – absorp., distrib., stor. – metab. proc. – analg., antipyret. – *CAAAL-0 A-1094.

In each of 6 trials (1 trial/week), 1 human subject ingested 200 g of 33.8% alcohol, i.e., $1.023 \text{ g} \pm 0.027 \text{ g/kg}$, within 1 min, plus aspirin (3.0-5.0 g) or pyramidon (0.9 g). The blood (Widmark method) and the urine were then analyzed. Psychological tests were also applied. The results showed that aspirin and pyramidon reduce and delay the absorption of alcohol, and delay its catabolism. The forensic implications of this fact are discussed, including, in particular, its application to traffic accidents. If the blood samples are taken a considerable time after ingestion, the blood alcohol value calculation for the time of the accident may show higher levels than are justified. This error can be avoided if a second blood sample is taken 90 min after the first, and the difference is taken into consideration.

1202. Schmidt, G.
FORENSISCH WICHTIGE FRAGEN DER BARBITURAT-AUSSCHIEDUNG IM HARN.
 [Questions of forensic importance on barbiturate elimination in the urine].
 Arzneimittelforschung (Aulendorf), 12(11): 1081-1085 (42 ref.), 1962.
 G – ES – SEC – general – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – other drug lev. – barbiturates – *CAAAL-0 A-1095.

The barbiturate excretion in urine is discussed, with special emphasis on forensic implications. If alcohol and a barbiturate have been both ingested, the time of maximal effect of each of the drugs must be considered. Different barbiturates have very different durations and peaks of action. Consequently, if 2 or more barbiturates have been administered, the maximal effects of each of them have to be considered. Synergistic effects may be expected, even after application of small doses, especially if the peak of action is reached at about the same time.

1203. Schneider, A.
BEURTEILUNG DER FAHRTÜCHTIGKEIT BEI DEM ZUSAMMENWIRKEN VON MEDIKAMENT UND ALKOHOL AUS DER RICHTERLICHEN SICHT. [Evaluation in court of driving ability in cases of interaction between drugs and alcohol].
 Blutalkohol (Hamburg), 2: 415-424 (15 ref.), 1964.
 G – general – DC (unspec.) – med.-leg. – mot. vehic. – humans – blood lev. – analg., antipyret. – tranquilizers – *CAAAL-0 A-1096.

Discussed are forensic problems arising from the use of alcohol and/or drugs by automobile drivers. The author tries to clarify: the difficulty of proving that drugs have been ingested, at what point a driver is impaired due to an alcohol-drug combination, and the problem of how intoxicated drivers can be legally convicted. A few court decisions are discussed. In one, the defendant was acquitted, although he had an alcohol blood level of $2^{\circ}/\text{oo}$, and had taken only "one or two" psyquil tablets. In a second case, the defendant was convicted, even though he had had a blood alcohol level of only $1^{\circ}/\text{oo}$, and had taken 8 "klar" and 10 aspirin tablets. The author, a judge, adds that it seems necessary that additional research should be undertaken, to permit a more uniform evaluation of the interaction of alcohol and drugs.

1204. Schoen, R.
BEITRÄGE ZUR PHARMAKOLOGIE DER KÖRPERSTELLUNG UND DER LABYRINTHREFLEXE. XXIII. MITTEILUNG: DIE ANTAGONISTISCHE BEEINFLUSSUNG DER NARKOSE DURCH ERREGUNGSMITTEL. [Contributions to the pharmacology of the body position and labyrinthal reflexes. XXIII: Antagonistic action of stimulants on narcosis].

Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 113: 275-304 (28 ref.), 1926.
 G – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – CNS – integ. syst. agents – stimulants – *CAAAL-0 A-1097.

Rabbits were used to study the excitatory effect of caffeine on the alcohol narcosis; of camphor on the alcohol and urethane narcoses; of hexeton on the alcohol, paraldehyde, and urethane narcoses; and of cardiazole on the paraldehyde narcosis. An antagonistic effect between caffeine and alcohol was observed only when all the righting reflexes (RR) were not paralyzed; the body RR and labyrinth reflexes were not restored, even when the caffeine doses were increased to produce spasms. In mild or medium alcohol narcoses, the administration of caffeine produced a short-term restoration of the RR. Administration of camphor iv had the same effect in alcohol and urethane narcoses. An antagonistic effect against all degrees of narcosis occurred with the administration of 3-12 mg iv of hexeton—10-20 mg produced spasms. Iv, sc, and stomachal administrations of cardiazole regularly produced antagonism against alcohol, urethane, and paraldehyde narcoses, with repeated injections achieving a lasting interruption of the narcosis.

1205. Schreiner, G. E., Berman, L. B., Kovach, R., and Bloomer, H. A.
ACUTE GLUTETHIMIDE (DORIDEN) POISONING: THE USE OF BEMEGRIDE (MEGIMIDE) AND HEMODIALYSIS
 Arch. Intern. Med. (Chicago), 101: 899-911 (29 ref.), 1958.
 E – SEC – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – humans – absorp., distrib., stor. – CNS – sed., hypnot. – *CAAAL-0 A-1352.

The article describes 6 suicide attempts by self-administered overdoses of glutethimide, 1 successful. 2 of the attempts, including the successful one, were complicated by previous alcohol ingestion, and another concerned possible alcohol intake. This is an expected pattern, because of the likelihood of alcohol drinking during deliberations of suicide. Alcohol has 2 possible effects on glutethimide. It may markedly enhance solubility of this drug, or it may increase CNS toxicity. Thus, it is difficult to tell whether alcohol has a synergistic effect on cerebral toxicity, or if it merely appears to have that effect because of the greatly accelerated glutethimide absorption. The author states that, “the problem of alcohol deserves the serious consideration of any physician prescribing glutethimide in a depressed patient.”

1206. Schroeter, L. C., and Zupko, A. G.
OBSERVATIONS ON THE METABOLISM OF ALCOHOL.
 J. Amer. Pharm. Ass. (Washington), 43: 270-272 (16 ref.), 1954.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – liver, kidney – metab. proc. – autocoids – autonomic agents – elect., water-bal. agents – sed., hypnot. – unclass. ther. agents – *CAAAL-7636-A2 A-1098.

In vivo and in vitro studies were conducted on alcohol metabolism. In 1 test, 7 rabbits received 0.75 g/kg 25% alcohol sol iv plus 1 g/kg fructose po or iv; 5 were given alcohol plus 5, 13, or 32 mg adenosine triphosphate (ATP); 4 received 50 mg chlortrimeton maleate (chlorprophenpyridamine) iv, either simultaneously with or 10 min prior to the alcohol, or 300 and 600 g im, 30 and 50 min before the alcohol, respectively; 4 were administered 5 or 10 mg probanthene (propantheline bromide) iv, 18 min before alcohol; and 2 received 10 mg dormison after ethanol. Only ATP and fructose accelerated alcohol metabolism. The mean decline of blood alcohol was between 19 and 25 mg/100 ml/hr in controls and in rabbits receiving chlortrimeton, probanthene, or dormison; 33 and 35 mg/100 ml/hr in the fructose rabbits, and 28, 30, and 37 mg/100 ml/hr in the ATP animals.

1207. Schultz, J. D., Rea, E. L., Fazekas, J. F., and Shea, J.
CHLORPROMAZINE IN THE MANAGEMENT OF ACUTE ALCOHOLIC STATES.
 Quart. J. Stud. Alcohol (New Haven), 16(2): 245-250 (8 ref.), 1955.
 E – exp. – DC (unspec.) – humans – drug-dep. humans – acute admin. – in vivo – dose resp. – blood lev. – mot. perform. – cardiovasc. – CNS – G.I. tract – tranquilizers – *CAAAL-6401-D1
 A-1099.

A clinical study of 164 acute alcoholic patients treated with chlorpromazine is reported. The drug was found extremely helpful in controlling motor excitement, nausea, and vomiting, and in producing relief from tension and anxiety. To test the reported synergism of chlorpromazine with alcohol, 4 normal subjects were given alcohol im in amounts sufficient to raise their blood levels to over 100 mg/100 ml. 100 mg of chlorpromazine were then given im, and all subjects went to sleep within 30 min. Despite the high blood alcohol level and the large dose of chlorpromazine, all patients could be easily aroused.

1208. Schüppel, R., and Soehring, K.
VERMEHRTE AMEISENSÄURE-AUSSCHIEDUNG—EIN MASS FÜR GESTEIGERTE DEMETHYLIERUNGSVORGÄNGE IN VIVO? [Increased formic acid excretion—a measure for heightened demethylation occurrences in vivo?].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 251(2): 109 (0 ref.), 1965.
 G – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – analg., antipyret. – *CAAAL-0
 B-0443.

To evaluate its suitability as a measuring value for the total metabolism of the animal, the daily formic acid excretion (DFAE) was determined in rats under the following conditions: after oral application of 150 mg/kg of amidopyrine, after amidopyrine plus phenobarbital stimulation, and after a parenteral application of 150 mg/kg of amidopyrine plus a massive dosage of alcohol (causing a blood alcohol level for 15 to 18 hr of 2.5-4°/oo. This method proved to be a relatively simple means of measuring the demethylation reaction in the animal. The DFAE curve after amidopyrine and alcohol can be explained by the inhibition of the N-demethylation.

1209. Schüppel, R.
EINFLUSS AKUTER ÄTHANOLEINWIRKUNG AUF DIE N-DEMETHYLIERUNG ZWEIER ARZNEIMITTEL BEI DER RATTE. [Influence of acute ethanol action on the N-demethylation of two drugs in the rat].
 Naunyn Schmiedeberg. Arch. Pharm. Exp. Path. (Berlin), 257(1): 60-61 (2 ref.), 1967.
 G – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – metab. proc. – *CAAAL-0
 B-0444.

Investigated was the influence of ethanol on the N-demethylation of amidopyrine-4-aminoantipyrene and acetylaminoantipyrene. Ethanol was found to cause a biphasic disturbance in the elimination of the main metabolites of the above drugs, the disturbance being indicated by an initial decrease and a following increase after catabolism of ethanol. The excretion of phenazone was not influenced by ethanol.

1210. Schüppel, R., Breyer, J., Streller, J., and Soehring, K.
WEITERE UNTERSUCHUNGEN ZUM ARZNEISTOFFWECHSEL DER RATTE UNTER ÄTHANOLBELASTUNG. [Further studies on drug metabolism in the rat under ethanol load].
 Naunyn Schmiedeberg. Arch. Pharm. Exp. Path. (Berlin), 257(3): 329-330 (4 ref.), 1967.
 G – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – in vitro – blood lev. – metab. proc. – analg., antipyret. – *CAAAL-0
 B-0445.

Investigated was the influence of alcohol on the C-hydroxylation of phenazone in the rat. Rats received ethanol (3.2 ml/kg) and phenazone (40 mg/kg). The phenazone excretion was, for the first 5 hr, 32% lower than in the water-phenazone controls. Alcohol also reduced the rate of metabolism of acetanilide in tests with in vitro liver slices. A considerably slower decline of the pentobarbital blood level (administered 20 mg/kg ip) was found after ethanol ingestion (blood level 1.6-2.2°/oo).

1211. Schüppel, R.

PHARMAKOKINETISCHE UNTERSUCHUNGEN AN DER ALKOHOLISIERTEN RATTE: EIN BEITRAG ZUR ANALYSE DER KOMBINATIONSWIRKUNGEN VON ÄTHANOL UND ARZNEIMITTELN. [Pharmacokinetic investigations on the alcoholized rat: a report on the analysis of the combined effects of ethanol and drugs].

Dissertation, Faculty of Mathematics and Natural Sciences, Eberhard-Karls University, Tübingen, West Germany, 137 pp. (65 ref.), 1968.

G – exp. cont. – exp. comp. – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – CNS – liver, kidney – metab. proc. – analg., antipyret. – anti-infectants – barbiturates – *CAAAL-0 B-1020.

The pharmacokinetic behaviour of certain drugs, under moderate alcohol influence, was investigated in rats (120-300 g). A series of 6 experiments was performed to study the effects of po administration of 3.0-3.3 ml/kg ethanol on: the N-demethylation of aminophenazone and phenazone, the C-hydroxylation of phenazone and pentobarbital, the acetylation of sulfanilamide and 4-aminoantipyrine, and the O-glucuronidation of 4-hydroxyphenazone. In another series, the effects of alcohol on blood levels of phenazone, pentobarbital, and 4-aminoantipyrine were studied. In addition, the effect of 3.0 ml/kg ethanol po on sleeping time induced by 20 mg/kg pentobarbital was determined. The concentration of metabolites formed through N-demethylation and C-hydroxylation was strongly decreased by ethanol, while the levels of metabolites formed through acetylation and O-glucuronidation were significantly increased. Ethanol significantly delayed elimination of phenazone and pentobarbital from the blood; however, after ethanol, the blood level of 4-aminoantipyrine reached only 1/2 of the control value. The effect of ethanol on pentobarbital sleeping time was supra-additive. It is concluded that ethanol reversibly inhibits drug metabolism, and that ethanol-induced disturbances in the pharmacodynamics can be particularly expected with drugs which are metabolized through an oxidative microsomal reaction, have a rapid metabolism, and of which the metabolites have a potency greater than that of the drug itself.

1212. Schüppel, R.

UNTERSUCHUNGEN ZUR SPEZIFITÄT ÄTHANOLBEDINGTER ARZNEISTOFFWECHSELSTÖRUNGEN. [Investigations on the specificity of ethanol-induced disturbances of drug metabolism].

Naunyn Schmiedeberg. Arch. Pharm. Exp. Path. (Berlin), 260 (2-3): 197-198 (4 ref.), 1968.

G – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – metab. proc. – analg., antipyret. – anti-infectants – *CAAAL-0 B-0446.

The effects of ethanol (3.2 ml/kg = max blood alcohol level, 2.2°/oo) on drug metabolism, with respect to the p-glucuronidation of 20 mg/kg of 4-hydroxy-phenazone, sulfanilamide acetylation (20 mg/kg), and the acetylation of 4-amino-antipyrine (20 mg/kg) were investigated in the rat. All 3 reactions were not directly disturbed, but certain oxidative side reactions in the metabolism of the drugs were transferred and influenced. The mechanism of action of ethanol on the metabolism of the drugs could not be clearly defined, but some interesting indications will be further investigated.

1213. Schüppel, R.

KONJUGATIONSREAKTIONEN IM ARZNEISTOFFWECHSEL DER RATTE BEI AKUTER ÄTHANOLBELASTUNG. [The influence of acute ethanol administration on drug

conjugation in the rat].

Naunyn Schmiedeberg Arch. Pharm. Exp. Path. (Berlin), 265: 233-243 (27 ref.), 1969.
G – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – metab. proc. – analg., antipyret. – anti-infectants – *CAAAL-0 B-0983.

The influence of ethanol (3.2 ml/kg 8-10% ethanol sol po) on drug N-acetylation, using sulfanilamide and 4-aminoantipyrine, and on drug O-glucuronidation, using norphenazone and 4-hydroxyphenazone, were studied in white male rats. All drugs were administered simultaneously with ethanol, in a dose of 20 mg/kg in 33 ml/kg water po. A control group received the drugs plus water. The urine levels of conjugates and of freely-eliminated unchanged substrates were determined up to 12 hr after administration. The excretion levels of the conjugates of sulfanilamide and norphenazone in urine were not affected by ethanol. There was, however, an 80% increase in excretion of the conjugates of 4-aminoantipyrine at a blood alcohol concentration of 1°/oo or more; 53% of the free and acetylated 4-aminoantipyrine was recovered from the urine, compared to 41% in controls. The same result was observed with 4-hydroxyphenazone, 83% of which was found in urine, compared with 67% in controls. Sulfanilamide and 4-hydroxyphenazone caused increases in diuresis of 29 and 28%, respectively. It is concluded that both N-acetylation and O-glucuronidation exhibit basically the same response, i.e., a shift from an oxidative to a predominantly conjugative metabolic pattern with drugs which have such alternative pathways, when under moderate alcohol influence in vivo. The results show a highly selective type of interaction between ethanol and the pharmacokinetics of drugs.

1214. Schüppel, R.

HEMMUNG DER HYDROXYLIERUNG VON ARZNEIMITTELN IN VIVO UNTER

ÄTHANOLBELASTUNG. [Inhibition of drug hydroxylation in vivo by ethanol administration].

Naunyn Schmiedeberg Arch. Pharm. Exp. Path. (Berlin), 265(2): 156-169 (49 ref.), 1969.
G – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – metab. proc. – analg., antipyret. – *CAAAL-0 B-0984.

The inhibition of the C-hydroxylation and N-demethylation of drugs by ethanol in vivo was investigated in white rats (120-140, and 200-400 g). The animals received phenazone (40 mg/kg po) or aminopyrine (20 mg/kg po or iv), either with an aqueous sol with a sol containing 3.2 ml/kg 95% ethanol po. Blood and urine samples were analyzed for phenazone and the metabolites, 4-aminoantipyrine, norphenazone, and 4-hydroxyphenazone. Blood alcohol concentrations (BAC) were also determined in a group given no drugs. Aminoantipyrine excretion was decreased by 66% at a BAC of 1-2.5°/oo; following total ethanol elimination, this value rose to 158% of the control. Ethanol caused a decrease of 57% in the max antipyrine blood level, and delayed the peak by 3 hr. Similar results were found with norphenazone and 4-hydroxyphenazone. Ethanol also delayed phenazone elimination from plasma, and increased excretion of unchanged phenazone by 22% in the first 5 hr; after the complete elimination of ethanol, this rate of increase rose to 43%. It is concluded that the inhibitions of N-demethylation and C-hydroxylation are similar, and are localized in the liver microsome oxidase.

1215. Schüppel, R.

PHARMACOKINETIC ASPECTS OF ETHANOL-DRUG INTERACTION.

In: *Alkohol und Verkehrssicherheit*: Konferenzbericht der 5. Internationalen Konferenz über Alkohol und Verkehrssicherheit. [Alcohol and Traffic Safety: proceedings of the 5th International Conference on Alcohol and Traffic Safety]. Freiburg im Breisgau, West Germany, 1969. Freiburg im Breisgau: Hans Ferdinand Schulz Verlag, pp. I.17-I.19 (4 ref.), 1969.
E – presentation – DC (add., infra-add., unspec. incr.) – mammals – blood lev. – CNS – liver, kidney – metab. proc. – analg., antipyret. – anesthetics – barbiturates – *CAAAL-0 B-0557.

The altered drug metabolism and changes in the pharmacokinetic behavior of drugs by ethanol-drug interactions are discussed. Hydroxylation of pentobarbital and phenazone, and N-demethylation of phenazone and aminopyrine are considerably diminished under the acute action of ethanol, resulting in a prolonged decrease in blood levels, and a derangement of the elimination pattern of typical metabolites in the urine. In vitro experiments by the author have shown that ethanol competitively inhibits microsomal N-demethylation by binding to the microsomal hydroxylating enzyme system. Certain reactions of drug metabolism, such as esterolysis or conjugations, are unaffected by ethanol. Drugs with a mixed metabolic elimination pattern show a depression of microsomal oxidation, while the other pathways are increased as compensation. Experiments on rats have shown that barbiturates, such as pentobarbital, produce an increased sleeping time and a prolonged decrease of blood levels, when administered with moderate doses of alcohols. Drugs such as barbital and thiopental, which are not metabolized by microsomal oxidation, are not affected by ethanol.

1216. Schüppel, R.

BIOCHEMICAL MECHANISMS OF HYPNOTIC DRUG-ETHANOL-INTERACTION.

Scand. J. Clin. Lab. Invest. (Oslo), 25 (Suppl. 113): 15 (0 ref.), 1970.
 E – abst. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – CNS – liver, kidney – metab. proc. – anesthetics – barbiturates – sed., hypnot. – *CAAAL-0 B-0985.

A series of narcotic and hypnotic drugs, comprising various barbiturates, thiopental, ketamine, propanidid, hydroxydione, clomethiazole, and 4-HO-butyric acid ethyl ester, were tested for prolongation of ethanol (1.5-4.5 ml/kg po) sleeping time in the rat. Pronounced prolongation of sleeping time occurred only with pentobarbital and ketamine, and a moderate prolongation was found with hexobarbital, phenobarbital, and clomethiazole; these 5 drugs were inactivated by microsomal hydroxylation. This effect was paralleled by a marked increase of the half-life of pentobarbital in rats pretreated with ethanol, and by competitive inhibition of microsomal N-demethylation of ketamine in vitro by ethanol. The failure of barbital, thiopental, hydroxydione, 4-HO-butyric acid ethyl ester, and propanidid to prolong ethanol narcosis, underlines a connection with previous findings that these drugs are inactivated by mechanisms other than drug hydroxylation (which is affected by ethanol). It is concluded that inhibition of microsomal hydroxylation by ethanol plays an important role in mechanisms related to drug-ethanol interactions.

1217. Schüppel, R.

MECHANISM OF HYPNOTIC DRUG-ETHANOL INTERACTION.

Industr. Med. Surg. (Chicago), 39(7): 309 (0 ref.), 1970.
 E – abst. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – CNS – liver, kidney – metab. proc. – anesthetics – barbiturates – sed., hypnot. – *CAAAL-0 B-0986.

Ethanol has been shown to inhibit microsomal hydroxylation, thereby interfering with the inactivation of drugs. This mechanism may favour a pharmacokinetic concept of ethanol-drug interactions. Experiments were conducted to determine the prolongation effect on ethanol sleeping time of different barbiturates, thiopental, ketamine, propanidid, hydroxydione, chlomethiazole, and 4-HO-butyric acid ethyl ester. The results confirmed the concept that the inhibition of microsomal hydroxylation by ethanol plays a highly selective part in mechanisms of ethanol-drug interaction.

1218. Schüppel, R., Zange, M., Dürr, W., and Petruch, F.

UNTERSUCHUNGEN ZUR WIRKUNGSINTERFERENZ VON KETAMINE (KETANEST) UND ÄTHANOL. [Ketamine-ethanol interaction: effects and mechanism].

Naunyn Schmiedeberg Arch. Pharm. Exp. Path. (Berlin), 266(4-5): 447 (0 ref.), 1970.
 G – exp. cont. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – CNS – liver, kidney – metab. proc. – anesthetics – *CAAAL-0 B-0987.

The interactions of alcohol, DDT, and phenobarbital with ketamine, a new anesthetic, were studied in female rats (125 and 250 g) and mice. In 1 experiment, rats were pretreated with an activator (1 x 100 mg/kg DDT) and an inhibitor of microsomal hydroxylation (3 x 80 mg/kg phenobarbital sodium), 7 days prior to administration of 65 or 80 mg/kg ketamine ip. The duration of ketamine narcosis was decreased to 30-50% of control values. In another experiment, 1.5, 3.0, or 4.5 ml/kg ethanol was given po to rats, 20 min prior to the above ketamine dose; the 3 ethanol doses increased the duration of narcosis by 200, 300, and 400%, respectively. A toxicity study with mice showed that the LD₅₀ of ketamine decreased from the control value of 275 mg/kg to 214 mg/kg with additional administration of 2.11 mg/g ethanol, and to 163 mg/kg with 4.22 mg/kg ethanol. It is concluded that ketamine metabolism is competitively inhibited by ethanol.

1219. Schuth, W.

SUIZIDVERSUCH MIT NEOTEBEN BEI GLEICHZEITIGEM ALKOHOLGENUSS.

[Attempted suicide with neoteben and simultaneous alcohol consumption].

Munchen. Med. Wschr. (Munich), 106(48): 2201-2203 (16 ref.),

1964.

G – general – case hist. – DC (add., infra-add., unspec. incr.) – humans – CNS – nerv. syst. – analeptics – anti-infectants – elect., water-bal. agents – tranquilizers – *CAAAL-0 A-1100.

A 37 yr-old man ingested 100 isonicotinic acid hydrazide (INH) tablets (0.1 g each), followed by 6 one-half-liter bottles of beer. The symptoms and treatment are described in detail. The patient was treated for 51 days, and was released free of symptoms. Since the same amount of INH without alcohol causes only mild neurological symptoms, the potentiating effect of alcohol is considered to be the reason for the severe effects.

1220. Schwarzmann, V., Infante, R., Petit, D., and Berthaux, N.

PRÉVENTION PAR L'ALCOOL ÉTHYLIQUE DES LÉSIONS HÉPATIQUES INDUITES PAR LE FORMIATE D'ALLYLE. [Prevention by ethyl alcohol of hepatic lesions induced by allyl formate].

Rev. Franc. Etud. Clin. Biol. (Paris), 14(10): 1014-1017 (10 ref.),

1969.

F – ES – exp. cont. – DC (decrease) – mammals – acute admin. – in vitro – liver, kidney – *CAAAL-0 B-0558.

Isolated rat livers were perfused with a blood-albumin mixture to which 0.04 ml allyl formate was added. 90 and 120 min later, histological examinations were made, and fluid samples taken and analyzed. It was found that lactic dehydrogenase, malic dehydrogenase glutamic oxaloacetic transaminase, and glutamic-pyruvic transaminase entered the perfusing medium, and severe liver injury resulted. When 1.10 g ethanol was added to the perfusing fluid prior to the allyl formate, damage to the liver was completely prevented. It is thought that ethanol blocked the active site of liver alcohol dehydrogenase, preventing it from acting on allyl alcohol, a derivative of allyl formate. This prevented the formation of acrolein, which is responsible for the damage and for the leakage of enzymes. A discussion of previous and related experiments is included.

1221. Schwedtke, G.

STUDIEN ÜBER METHÄMOGLOBINBILDUNG. IX. MITTEILUNG: EINFLUSS DES ALKOHOLS AUF DIE METHÄMOGLOBINBILDUNG DURCH ANILIN. [Studies on methemoglobin formation. IX. Influence of alcohol on the formation of methemoglobin by aniline].

Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 188: 130-137 (11 ref.),

1938.

G – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – dose resp. – blood lev. – blood comp., sites, lymph – indust. intox. – *CAAAL-0

A-1458.

The LD₅₀ of aniline was determined in mice, then 17 mice in 3 groups (5, 6, 6) received 0.6, 2.0, or 5.0 mg/g alcohol po, shortly before administration of 3.0 mg/g aniline sc. Another 18 mice in 3 groups (6, 6, 6) received 1.0, 2.0, or 4.0 mg/g alcohol po after administration of 0.36 mg/g aniline sc. Similarly, another 10 mice (5,5) received 0.36 mg/g *p*-aminophenol sc, followed by 4.0 mg/g alcohol po or sc. Cats were given 1 g/kg 20% alcohol po/day or 1 g/kg 104% alcohol sc/day for 14 days, followed, after a 2-day pause at the end of administration, by 2.0 mg/kg aniline sc. Alcohol increased the effect of aniline, as shown by a blood methemoglobin content which was 1 1/2 times higher than normal. The minimal effective dose of aniline was decreased by 20 times (from 2 mg/kg to 0.1 mg/kg). The *p*-aminophenol experiments showed similar results, as did the cat experiments with chronic alcohol administration. It is concluded that the combined effect of alcohol and aniline may be additive, and is due to an inhibition of methemoglobin reduction by alcohol.

1222. Scogin, J. T., and Dobson, H. L.
 ACUTE ALCOHOLIC INTOXICATION: USE OF METHYLPHENIDYLATE.
 American Practitioner (Philadelphia), 11(1): 48-51 (9 ref.), 1960.
 E – exp. – DC (antidotal) – humans – cardiovasc. – CNS – stimulants – *CAAAL-9252-N13
 A-1101.

50 acutely-intoxicated patients with depressions of various intensities, received 30 mg (17 persons), 50 mg (25), 50-100 mg (5), or 100-130 mg (3) of methylphenidylate iv; amounts of more than 50 mg were given in multiple doses. After a latent period of 3-10 min, a usually dramatic change was made to a more alert, responsive, and sober state which was maintained with a slow improvement. Only in 2 cases was no beneficial response noted; both agitated patients became more aggressive and pugnacious. Some side effects were noted. The recommended dosage, with max results and min side effects, is 20-30 mg iv initially, followed by the same dose every 1/2 hr until improvement is shown; only in the completely unresponsive alcoholic should 50 mg be given in 1 injection.

1223. Scott, P. D.
 OFFENDERS, DRUNKENNESS AND MURDER.
 Brit. J. Addict. (London), 63: 221-226 (6 ref.), 1968.
 E – SEC – stat. surv. – conj. addict. – DC (add., infra-add., unspec. incr.) – med.-leg. – humans – drug-dep. humans – tranquilizers – *CAAAL-13572
 B-0559.

A psychiatrist reports on the incidence of alcoholic intoxication and drug involvement in 50 consecutive, personally-examined murder cases. The murderers ranged in age from 13-72 yr of age, and, except for 4 women aged 21, 31, 32, and 57 yr, were males. Among the murderers, 4 were chronic alcoholics, of which 1 was conjunctively addicted to librium and barbiturates. This last case was a 32-yr old mother who killed her child while suffering from a depressive psychosis. In an analysis of the use of alcohol at the "material time" of the offences, it was determined that 11 cases had been drinking shortly before the offence, 8 of them heavily. In 1 of these 11 cases, the action of alcohol was potentiated by librium taken on prescription, and, in another case, by librium plus barbiturates (taken without authorization). Of the 21 teenagers (all male) in this series, 3 had been drinking at the time of the offence, and 3 others were amphetamine users.

1224. Sebastianelli, A.
 L'AZIONE DEL DEIDROCOLATO SODICO SULLA ALCOOLEMIA PROVOCATA IN SOGGETTI NORMALI. [The effect of sodium dihydrocholine on the blood alcohol level in normal subjects].
 Arch. Fisiol. (Florence), 38: 26-35 (9 ref.), 1938.
 I – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – liver, kidney – metab. proc. – *CAAAL-0
 A-1102.

The influence of sodium dihydrocholine (10 cc iv in a 20% sol) on the blood alcohol level was determined in a series of controlled experiments with 7 subjects (5 normal and 2 hepatic patients), who were first given 0.5 cc/kg 95% alcohol in a 20% aqueous sol po. The blood alcohol curve was determined with Widmark's modified micro-method. In 4 of the 5 normal subjects, sodium dihydrocholine reduced the blood alcohol concentration (22 to 49%). The results lead to the conclusion that the substance in question accelerates the combustion of alcohol in normal subjects, and that the reduction in the blood alcohol level is probably due to an excitation of the liver function, with respect to alcohol metabolism.

1225. Seeberg, V. P., and Dille, J. M.

THE COMPARATIVE RATE OF GASTROINTESTINAL ABSORPTION OF BARBITAL, SODIUM BARBITAL AND ELIXIR OF BARBITAL N. F. VII.

J. Amer. Pharm. Ass. (Washington), 32: 133-137 (6 ref.), 1943.
E – SEC – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – CNS – G.I. tract – barbiturates – *CAAAL-0 A-1103.

The differences in the rate of absorption between different pharmaceutical preparations were studied in cats. The blood and the contents of the stomach, intestine, and colon were assayed separately for barbitol. The procedures for the determination of absorption and the method of assaying are described. The depression after the administration of the elixir was proportionally greater than would be expected from the blood level of the barbitol, and this was considered to be due to the effect of 2.7 cc of alcohol/kg in the elixir.

1226. Seevers, M. H.

AMPHETAMINE AND ALCOHOL (QUESTIONS AND ANSWERS).

J.A.M.A. (Chicago), 184(10): 843 (0 ref.), 1963.
E – general – DC (add., infra-add., unspec. incr.) – humans – CNS – amphetamines – *CAAAL-10391-D3 A-1104.

In reply to the question, "What might be expected regarding changes in human behaviour from the simultaneous ingestion of ethyl alcohol and amphetamine sulfate?" it is stated that, pharmacologically, the depressant effects of alcohol would be antagonized. This could prolong, rather than prevent, the "excitement" state and the duration of abnormal behaviour. Large quantities of amphetamines, as might be ingested in heavy chronic alcoholism, could contribute to the hallucinogenic, excitatory, and other characteristics of delirium tremens. The abuse of large amounts of amphetamines with alcohol would worsen, rather than correct, the situation.

1227. Seidel, G., Streller, I., and Soehring, K.

DER EINFLUSS SUBCHRONISCHER ALKOHOLGABEN AUF DIE

PENTOBARBITALAUFNAHME DES MEERSCHWEINCHENHIRNS. [The influence of subchronical alcohol doses on the intake of pentobarbital in the brain of the guinea pig].

Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 247(4): 312-313 (1 ref.), 1964.
G – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – absorp., distrib., stor. – CNS – barbiturates – *CAAAL-0 A-1105.

Pentobarbital, 20 mg/kg ip, was administered to guinea pigs which, for 1 week, had been given a 5% alcohol sol instead of water. 30 min after injection, the serum level was $22.1 \pm 7.0 \mu\text{g/ml}$ ($P = 0.02$), and $15.4 \pm 5.6 \mu\text{g/ml}$ in the controls; after 110 min, the level was $15.2 \pm 4.8 \mu\text{g/ml}$ ($P = 0.2$), and $13.3 \pm 2.5 \mu\text{g/ml}$ in the controls. Apparently the ethanol impedes the transport of pentobarbital into the CNS.

1228. Seidel, G., Streller, I., and Soehring, K.
 ZUR FRAGE DER BEEINFLUSSUNG DES ALKOHOL-GEHALTES IM BLUT DURCH
 CHLORPROMAZIN. [On the effect of chlorpromazine on blood alcohol concentration].
 Arzneimittelforschung (Aulendorf), 14: 412-415 (14 ref.), 1964.
 G – ES – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – blood
 lev. – metab. proc. – tranquilizers – *CAAAL-11338-A2 A-0358.

After premedication of 9 fasting dogs with sc and oral doses of 5 mg/kg chlorpromazine, the absorption and elimination of alcohol in doses of 0.79 and 1.58 g/kg remained unaffected. Following absorption of alcohol, even iv administration of chlorpromazine did not affect alcohol elimination. Indications are given of the variable fate of alcohol in the system of the same dog. Reports in which the blood-alcohol content and breakdown of alcohol in the blood are claimed to be influenced by chlorpromazine are discussed.

1229. Seidel, G., and Soehring, K.
 ZUR FRAGE DER ÄNDERUNG DER BLUTALKOHOLWERTE DURCH
 MEDIKAMENTE. [The question of drug influence on blood alcohol levels].
 Arzneimittelforschung (Aulendorf), 15: 472-474 (19 ref.), 1965.
 G – ES – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – metab.
 proc. – analeptics – analg., antipyret. – anti-infectants – sed., hypnot. – tranquilizers –
 *CAAAL-11912-A2 B-0447.

41 commonly-used drugs were administered to 41 dogs (wt—10 to 20 kg). Immediately afterwards, the dogs received 0.79 g/kg ethanol in a 20% sol by oesophageal tube. Blood alcohol levels were tested for the next 6 hr. The distribution range was the same as that of the controls. Neither acceleration nor retardation of the alcohol elimination was observed. The dosages and the route of administration of the 41 drugs are tabulated.

1230. Seidel, G., Streller, I., and Soehring, K.
 BEEINFLUSSUNG DES BLUTALKOHOLWERTES DURCH ARZNEIMITTEL?
 [Influencing the blood alcohol level by drugs?].
 Aktuelle Probleme der Verkehrsmedizin (Stuttgart), 2: 148-151 (4 ref.), 1965.
 G – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – analg., antipyret.
 – barbiturates – hormones, hormone antag. – stimulants – tranquilizers – *CAAAL-0 B-0448.

Alcohol in a dose of 0.79 g/kg was given po. Bayer E 39, luminal, nicotoben, pyramidon, rastinon, reactivan, serpasil, and zentronal were administered 60 min after ethanol. Doses and route of administration are tabulated. The blood alcohol level and the curve of elimination were not influenced by the drugs.

1231. Seidel, G.
 VERTEILUNG VON PENTOBARBITAL, BARBITAL UND THIOPENTAL UNTER
 ÄTHANOL. [Distribution of pentobarbital, barbital, and thiopental under ethanol].
 Naunyn Schmiedeberg. Arch. Pharm. Exp. Path. (Berlin), 257(2): 221-229 (29 ref.), 1967.
 G – ES – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals
 – acute admin. – in vivo – blood lev. – other drug lev. – CNS – liver, kidney – skel., muscle, skin
 – barbiturates – sed., hypnot. – *CAAAL-12607-B2 B-0449.

White mice were divided into 3 groups. Group 1 received 60 mg/kg sodium pentobarbital ip 30 min after 3 g/kg ethanol po, or 60 mg/kg pentobarbital ip 20 min before ethanol (3 g/kg ip). Group 2 was given 250 mg/kg sodium barbital ip, 30 min after 3 g/kg ethanol po. Group 3 received 30 mg/kg sodium thiopental ip, 30 min after 3 g/kg alcohol po. It was found that the concentration of

pentobarbital in the liver, kidney, brain, muscle, and blood was higher and declined more slowly in mice treated with ethanol. The barbitol concentration was slightly increased in the liver and kidney after ethanol treatment, but was unaltered in the brain, muscle, and blood. The elevation of the concentration of thiopental in the liver, kidney, and brain after ethanol treatment was not statistically significant, and the concentrations in the muscle and blood were not altered. The decline of thiopental concentration was not affected by ethanol. It is considered that the elevation of the concentration of pentobarbital in the brain of mice treated with ethanol may explain in part the fact that pentobarbital anesthesia is deepened and prolonged by ethanol (as a result of an impaired degradation of pentobarbital).

1232. Sellschopp, U.

DIE ALIMENTÄRE ESSIGESTERKURVE IM UNBEHANDELTEN, ALKALISIERTEN UND ANGESÄUERTEM BLUT, IHRE BEEINFLUSSUNG DURCH HORMONE (INSULIN, THYROXIN, ADRENALIN, HYPOPHYSIN, PRAELOBAN), DURCH HORMONE UND GLUKOSE UND DURCH GENUSSGIFTE (NIKOTIN, COFFEIN): EIN BEITRAG ZUR FRAGE DES EXOGENEN UND ENDOGENEN BLUTALKOHOLS. [The alimentary acetic ester curve in untreated, alkalized, and acidified blood—the influence of hormones (insulin, thyroxin, adrenalin, hypophysin, prelobane), of hormones and glucose, and of mild poisons (nicotine, caffeine): a contribution to the question of exogenous and endogenous blood alcohol].

Dissertation, Hygienic Institute of the Friedrich Wilhelms University, Berlin, Germany, 28 pp. (26 ref.), 1940.

G – SEC – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – blood lev. – mot. perform. – metab. proc. – nutritive agents – stimulants – *CAAAL-0 A-1353.

Rabbits were given .01, .02, .025, or .03 moles ethyl acetate/kg body wt po, and the following were determined: the blood ester curves and their sodium hydroxide- and sulfuric acid-splitting curves; the changes in these curves by administration of adrenalin, thyroxin, insulin, hypophysin, prelobane, and insulin with zinc chloride, nicotine, and caffeine; the changes in the ester and ester plus hormone curves by administration of glucose; and the body temperatures of the animals. Among other results, it was found that there was an adaptation to the ester in chronic administration. Adrenalin and thyroxin lowered the blood ester level (BEL) in small doses, but raised it in large doses; this effect of the hormones was antagonized by glucose. Nicotine raised, and caffeine lowered, the BEL. The author concludes that the absorption and elimination of alcohol is dependent upon the hormone system, and can be altered by glucose, nicotine, and caffeine. The BEL also contributes to the degree of intoxication by alcohol—the esterification of alcohol occurs in the sense of a Cannizzaro reaction; it is closely tied to the metabolism of sugar, and is not a simple oxidation reaction.

1233. Serianni, E., and Baglioni, S.

L'AZIONE DELLA MORFINA SULLA CURVA ALCOOLEMICA PROVOCATA NELL'UOMO NORMALE. [The action of morphine on the blood alcohol curve in normal man].

Reale Accademia Nazionale dei Lincei, Atti. Classe di Scienze Fisiche, Matematiche, e Naturali, Rendiconti (Rome), (Ser. 6) 24: 485-487 (10 ref.), 1936.

I – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – analg., antipyret. – *CAAAL-0 A-1106.

3 human subjects were given injections of 33-35 cc of alcohol, with or without 1 cg morphine hydrochloride 25 or 26 min after the alcohol. The blood alcohol curves were plotted. It was found that morphine induces a more or less marked decline of the blood alcohol curve. The degree of decline of the curve in the subjects was 40%, 20%, and 30%, respectively.

1234. Serianni, E., Lolli, G., and Venturini, M.

THE EFFECTS OF SOLID FOOD AND OF ALCOHOLIC BEVERAGES, ESPECIALLY WINE, ON THE EXCRETION OF HIPPURIC ACID.

Quart. J. Stud. Alcohol (New Haven), 16: 67-85 (32 ref.),

1955.

E – exp. comp. – congen. stud. – humans – acute admin. – in vivo – other drug lev. – metab. proc.

– *CAAAL-6965-D1

A-1442.

To evaluate the effect of a meal, with or without wine, on hyperhippuricuria, 7 fasted, healthy human subjects (20-30 yr) received, on different occasions, no meal, a test meal, or a test meal with 12% red wine; sodium benzoate (6 g in 60 cc water) was given 1 hr after the end of the meal, and urine was then collected every 2 hr for 6-10 hr. In a second experiment, to study the effect on hyperhippuricuria of an aqueous ethanol sol or wine, 4 fasted subjects received, on different occasions, no alcohol, a 20% sol containing 0.5 cc ethanol/kg, or 12% red wine containing a corresponding amount of ethanol; 1 hr later, sodium benzoate was given, and urine collected at 2-hr intervals. It was found that sodium benzoate caused a higher excretion of hippuric acid after a meal than on an empty stomach. Wine ingested on an empty stomach did not substantially affect the total amount of hippuric acid excreted, but it did delay and prolong the excretion. Ethanol definitely decreased the total amount of hippuric acid excreted, and also delayed and prolonged the excretion. Wine with a meal caused a marked increase in hippuric acid excretion, as well as an earlier and more prolonged period of hyperhippuricuria.

1235. Serrano, P., and Chavez, R.

CLINICOTHERAPEUTIC EVALUATION OF CHLORPROPAMIDE, A NEW SULFONYLUREA DERIVATIVE FOR THE TREATMENT OF DIABETES MELLITUS.

Ann. N.Y. Acad. Sci. (New York), 74: 931-935 (19 ref.),

1958-59.

E – SEC – general – DC (sensit.) – humans – cardiovasc. – glands – hormones, hormone antag. –

*CAAAL-0

A-1316.

To confirm pharmacological findings that chlorpropamide has a higher potency than other sulfonylurea derivatives, i.e., carbutamide and tolbutamide, a clinicotherapeutic study was made on 33 Mexican labourers, all of whom had uncomplicated diabetes and were not alcoholic. Excellent or satisfactory results were obtained in 28 cases of moderate or slight diabetes, satisfactory results were gained in 2 cases of severe diabetes, and unsatisfactory results occurred in 3 cases with severe diabetes. Toxic effects were similar to other anti-diabetics, and the potency was 2-3 times that of carbutamide or tolbutamide. In 3 patients, chlorpropamide induced a generalized peripheral vasodilation, especially over the face and neck, for 2-4 hr following ingestion of a cup of wine or a glass of beer. The reaction was of variable intensity, and was not accompanied by hypertension or headache. A test was made with other patients, but the effect could not be reproduced.

1236. Sfrikakis, P.

ISONIAZID AND ALCOHOL.

Amer. Rev. Resp. Dis. (Baltimore), 101(6): 991 (1 ref.),

1970.

E – general – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – absorp., distrib.,

stor. – CNS – liver, kidney – metab. proc. – anti-infectants – *CAAAL-0

B-0988.

The author comments on a published report concerning the attempted treatment of a case of suicide resulting from intake of a massive dose of isoniazid (300 x 100 mg), and suggests that the concomitant ingestion of a bottle of wine may have been a significant factor. A similar case, treated by the author, concerned a suicide committed by a 40-yr-old male chronic alcoholic, who was being treated with isoniazid for pulmonary tuberculosis. The patient ingested 3 g of the drug, plus a large quantity of wine. Upon arrival at hospital 4 hr later, the man was deeply confused, and suffered generalized seizures every 5-10 min. 2 hr later, continuous status epilepticus developed, and he became cyanotic. Despite all forms of treatment (iv fluids, phenobarbital, scopolamine hydrobromide and chloral

hydrate), the patient died 7 hr after the isoniazid-alcohol intake. Although the dose of isoniazid was in comparison with the massive dose mentioned above, and although the patient treated by the author was an alcoholic, whereas the other suicide was not, it is the author's opinion that alcohol precipitated death in both cases. Experimental evidence, however, on the exact relationship of alcohol to isoniazid absorption and neural damage, or the exact mechanisms of actions, is lacking.

1237. Shagass, C., and Jones, A. L.

A NEUROPHYSIOLOGICAL STUDY OF PSYCHIATRIC PATIENTS WITH ALCOHOLISM.

Quart. J. Stud. Alcohol (New Haven), 18: 171-182 (14 ref.), 1957.
E – exp. cont. – cross-tol. – drug-dep. humans – chronic admin. – in vivo – psychol. perform. – CNS – barbiturates – *CAAAL-8527-E1 A-1443.

Sedation threshold (the amount of iv sodium amytal required to produce certain EEG and speech changes) findings, liver function test results, psychological test results, and psychiatric data were studied in 24 male and 16 female alcoholics, and were compared to similar data based on 290 non-alcoholic patients hospitalized for psychiatric treatment. It was found that the distributions of sedation thresholds were about the same in both groups. Signs of organic impairment, seen in 40% of the alcoholics, were associated with lower sedation thresholds. The greater the degree of dysphoria, the greater the sedation threshold. Patients hospitalized mainly because of external pressures had lower thresholds than patients hospitalized due to internal pressures. It is concluded that the sedation threshold findings in psychiatric alcoholic patients were no different than those in non-alcoholic patients. The psychological factors correlated with the threshold were the same in alcoholics as in non-alcoholics. No evidence was obtained of increased tolerance to barbiturates in association with alcoholism.

1238. Shelley, W. B.

GOLF-COURSE DERMATITIS DUE TO THIRAM FUNGICIDE: CROSS-HAZARDS OF ALCOHOL, DISULFIRAM, AND RUBBER.

J.A.M.A. (Chicago), 188(5): 415-417 (17 ref.), 1964.
E – SEC – general – case hist. – DC (sensit.) – humans – cardiovasc. – skel., muscle, skin – unclass. ther. agents – *CAAAL-11169-C1 A-1317.

A case of dermatitis, following exposure to a thiram fungicide, is described, and the general problem is discussed. The patient, a 51 yr-old male, was found to be extremely sensitive to the fungicide used on a golf course, and exhibited a severe, chronic, fissured, erythematous, scaling eruption of the hands, forearms, neck, face, and legs; the chest showed mummular patches of eczematous change. A rare allergic reaction such as this may also be seen as a pharmacological sensitization reaction after alcohol ingestion, following exposure to thiram. The intolerance reaction is quite possible outside of alcoholism therapy, since thiram is used as a catalyst in the vulcanization of rubber, and body moisture and sweat can leach the substance, under certain circumstances, from all such processed rubber products. A powerful fungicide, thiram is sprayed and dusted on farmlands and lawns, and on seeds, plants, fruits, and vegetables. Contact may also arise through the use of the substance as a larvacide, pesticide, and insecticide, or through its use as a bactericide in germicidal soaps or sprays.

1239. Shepherd, M.

CLINICALLY IMPORTANT EXAMPLES OF DRUG INTERACTION. PSYCHOTROPIC DRUGS (1) INTERACTION BETWEEN CENTRALLY ACTING DRUGS IN MAN: SOME GENERAL CONSIDERATIONS.

Proc. Roy. Soc. Med. (London), 58(11): 964-967 (20 ref.), 1965.
E – SEC – general – DC (add., infra-add., unspec. incr.) – humans – CNS – nerv. syst. – senses – analg., antipyret. – anti-infectants – autocoids – barbiturates – *CAAAL-0 B-0450.

Clinical studies of alcohol-barbiturate interactions are mentioned. Sudden withdrawal of large doses of either alcohol or barbiturate are said to cause the so-called abstinence syndrome, consisting of weakness, tremulousness, anorexia, anxiety, insomnia, convulsions, and delirium. A partial equivalence between alcohol and barbiturates was demonstrated by substituting one drug for another. It is felt that a number of drugs, including insoniazid, amidopyrine, the antihistamines, the opiates, the urea derivatives, and the sulphonamides can intensify the noxious effects of alcohol and adversely affect driving ability; a clear demonstration of such effects on human behaviour, however, depends on the active collaboration of psychologists who are prepared both to work experimentally with human subjects, and to pay due regard to pharmacological principles.

1240. Shimazu, R.

JODNATRIUM NO HIKAKYŪSHŪ WA SONO CHŪSHA-EKI NI KYŌZON SERU ALKOHOL, ADRENALIN OYOBİ NATRIUMNITRIT NI YORITE IKA NI EIKYŌ SERARURU KA. [How does alcohol, adrenaline or sodium nitrite influence the subcutaneous absorption of sodium iodide, when these drugs are added to a sodium iodide solution?].

Folia Pharmacol. Jap. (Nippon Yakurigaku Zasshi) (Kyoto), 23: 175-181 (7 ref.), 1937.
J – GS – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – other drug lev. – absorp., distrib., stor. – cardiovasc. – skel., muscle, skin – diagnost. agents – *CAAAL-0 A-1107.

Rabbits were injected with 0.5 cc/kg of a 2% sodium iodide sol which contained 2% or 20% alcohol, adrenaline hydrochloride (0.002%), or sodium nitrite (5%). To test the influence of the above drugs on iodine absorption, the iodine excretion in the urine was determined. Results showed that the addition of adrenaline impeded the sodium iodide absorption, due to a strong blood vessel contraction at the puncture. The 2% alcohol addition increased, and the 20% alcohol decreased, the absorption. The favourable influence of the alcohol is caused by a local hyperemia (due to a dilatation of blood vessels); the 20% alcohol caused an inflammation around the puncture which hampered the absorption. The vasodilatatory power of sodium nitrite favoured the absorption of sodium iodide.

1241. Shimazu, R.

NATRIUMSALICYLAT NO KINNIKUNAI KYŪSHŪ WA SONO CHŪSHA-EKI NI KYŌZON SERU ALKOHOL, ADRENALIN OYOBİ GUMMI ARABICUM NI YORITE IKA NI EIKYŌ SERARURU KA. [How does alcohol, adrenaline, or gum arabic influence the intramuscular absorption of sodium salicylate, when these drugs are added to a sodium salicylate solution?].

Folia Pharmacol. Jap. (Nippon Yakurigaku Zasshi) (Kyoto), 24: 67-72 (1 ref.), 1937.
J – GS – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – absorp., distrib., stor. – cardiovasc. – skel., muscle, skin – *CAAAL-0 A-1108.

Rats were injected in the musculus rectus femoris with 1.0 cc/kg of a 10% sodium salicylate sol, containing alcohol (2% or 20%), adrenaline hydrochloride (0.002%), or gum arabic (2% or 20%). The blood salicylate concentration was determined. The results showed that the 2% alcohol increased, and the 20% alcohol decreased, salicylate absorption to a significant degree. The beneficial effect of the alcohol on the salicylate absorption is mainly to be found in the fact that alcohol dilates the blood vessels, and thus produces local hyperemia. Concentrated alcohol, on the other hand, leads to extreme inflammation at the place of injection, which, as a result of the blood congestion which thereby occurs, disturbs the salicylate absorption. Both adrenaline and gum arabic decreased salicylate absorption.

1242. Shinaberger, J. H.

TREATMENT OF METHANOL POISONING BY EXTRACORPOREAL DIALYSIS.
Arch. Int. Med. (Chicago), 108: 937-939 (9 ref.), 1961.

E – SEC – general – case hist. – DC (antidotal) – DC (decrease) – humans – acid-base, blood pH, elect. – alcohols – elect., water-bal. agents – *CAAAL-10210-N8 A-1109.

An advanced case of methanol poisoning and acidosis in a 42 yr-old female patient was treated with a combination of alkali, ethanol, and extracorporeal dialysis. Prior to hemodialysis, 500 ml dextran plus 1,000 cc 5% dextrose in distilled water containing 60 cc 95% ethanol, 88 m equivalents sodium bicarbonate, and an additional 25 g dextrose were administered rapidly iv. An infusion containing 50 mg metaraminol bitartrate/l was initially given to relieve hypotension. A sustaining infusion of ethanol in 5% dextrose was administered throughout the dialysis at an average rate of 6 cc ethanol/hr. The patient made a surprisingly complete recovery, which was attributed to 2 factors: (1) although she was treated late in the course of the acidosis and had ingested large amounts of methanol, the preparation ingested contained ethanol, which protected her during the drinking spree and delayed the onset of acidosis, and (2) the rapid removal of methanol and its oxidative metabolites once they had reached toxic levels was achieved by extracorporeal dialysis.

1243. Shorell, I. D.

NEW VIEWS ON ALCOHOLIC BEVERAGES IN HEALTH AND DISEASE: MEDICAL ASPECTS OF COMPARATIVE TOXICITIES BASED ON CONGENERIC CONTENT.

Medical Record (New York), 147: 145-148 (25 ref.),

1938.

E – SEC – exp. comp. – general – congen. stud. – humans – mammals – other org. – CNS – G.I. tract – metab. proc. – skel., muscle, skin – indust. intox. – *CAAAL-0 A-1110.

The major emphasis in this paper is on the medicinal properties of whiskey. The results of research and observations by the author have led him to conclude that the "limited use of wholesome 'light' whiskies, such as a blend of mature whiskies with neutral spirits which is wholly purified and with nearly all injurious congeners eliminated, can be safely advocated by the physician for all patients who insists on indulgence in liquor, or who require a safe alcoholic stimulant. Among the briefly-mentioned investigations of the author, the comparative toxic properties of ethyl alcohol, various blended whiskies, and straight whiskies were studied with respect to their growth-inhibiting influence on a culture of *Staphylococcus aureus*, of standardized resistance to phenol. His clinical observations with patients were upheld by the experimental result that, "the alcohol diluted to one hundred proof and the blended whiskies [were found] to be essentially nontoxic in the sense of not exhibiting growth-inhibitory powers, and the straight whiskies to be definitely toxic in the sense that they prevented, or at least delayed, reproductive growth."

1244. Siegmund, B.

KÖNNEN DIE ERGEBNISSE DER WIDMARKSCHEN BLUTALKOHOLUNTERSUCHUNG DURCH MEDIKAMENTE BEEINFLUSST WERDEN? II. MITTEILUNG: PERVITIN. [Can the results of Widmark's blood alcohol determination be influenced by medicaments? II: Pervitin].

Deutsch. Med. Wschr. (Stuttgart), 65(19): 754-756 (6 ref.),

1939.

G – exp. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – CNS – amphetamines – *CAAAL-1165-A1 A-1111.

2 young healthy volunteers drank 31% cognac in a dose of 1 g/kg within 10 min. In one test, rings had to be put on a bar. In a second experiment, the subjects drank the same alcohol dose, and received in addition 12 mg pervitin po. Subjects A and B (subject B in parentheses) made, 90 (30) min after alcohol application, 7 (7) mistakes, and, 60 (30) min after alcohol followed by pervitin, 3 (8) mistakes. 60 (30) min after alcohol and pervitin simultaneously, 4 (1) mistakes were made. The norm, without any drug, was 1.6 (1.72) mistakes. It is concluded that the blood alcohol levels were not influenced by pervitin.

1245. Siepmann, H.

ÜBER DIE WIRKUNG VON CORAMIN AUF DIE BLUTALKOHOLKONZENTRATION UND DEN RAUSCHZUSTAND. [The action of coramine on blood alcohol concentration and the degree of drunkenness].

Med. Klin. (Munich), 34: 1192-1193 (6 ref.),

1938.

G – exp. cont. – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – CNS – respir. – stimulants – *CAAAL-876-A2 A-1112.

In experiments with rabbits, the animals received, in the first experiment, 3 cc/kg ethanol in 35 cc distilled water; 70 min later, 0.15 cc coramine was given iv. After 5 hr, the blood alcohol level was 1.0°/oo, and 1.5°/oo in the controls. Coramine caused a faster decline of the curve. In a second experiment, 5 cc/kg ethanol, and, 65 min later, 0.2 cc coramine iv were given. The coramine curve declined faster. In a third and fourth experiment with 5 cc/kg ethanol (5 cc/kg) and 0.2 cc coramine (3 times 0.2 cc), the effects were similar. It is concluded that coramine does not significantly influence the blood alcohol curve. The stimulating effect was temporary at the peak of the alcohol curve, and permanent in the declining state. When the rabbits were in a state of shock, coramine always terminated the shock.

1246. Siepmann, H.

CORAMIN UND BLUTALKOHOL. [Coramine and blood alcohol].

Dissertation, Medical Faculty of Philipps University, Marburg, Germany, 15 pp. (37 ref.), 1939.

G – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – blood lev. – cardiovasc. – CNS – respir. – stimulants – *CAAAL-0 A-1459.

In 1 series of experiments, rabbits received po 3 cc absolute alcohol/kg in 35 cc distilled water or 5 cc alcohol/kg in 50 cc water. Blood tests were performed every min up to 6 hr. 8 days later, the same animals received identical alcohol doses, followed by iv injections of coramine (0.15, 0.2, or 3 x 0.2 cc/kg), and blood tests were again made. In another series, 5.5 or 6.5 cc/kg alcohol was administered iv to rabbits, and 0.2 cc coramine was injected iv 3 times—during the ascending, at the peak, and during the descending blood alcohol curve (BAC). Coramine did not significantly alter the BAC, but it did have pronounced effects on intoxication. When given at the peak of the BAC, the awakening or sobering effects of coramine lasted only 15-20 min before intoxication symptoms reappeared. When given during the BAC decline, however, coramine had a more long-lasting stimulating effect. With higher alcohol doses, or when given during the rise of the BAC, coramine had no apparent stimulating effect. In every experiment, an increase of the respiratory rate was observed. It is concluded that, although the BAC is not affected, coramine does have sobering effects when administered in therapeutic doses.

1247. Sigg, E. B.

NEUROPHARMACOLOGIC ASSESSMENT OF TOFRANIL (IMIPRAMINE), A NEW ANTIDEPRESSANT AGENT.

Fed. Proc. (Bethesda), 18: 144 (0 ref.),

1959.

E – SEC – abst. – exp. cont. – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – CNS – antidepressants – *CAAAL-0 A-1113.

Parenteral administration of 1-5 mg/kg tofranil (imipramine) failed to have a stimulating effect in animals, and higher doses had a sedative effect. In contrast to chlorpromazine, the conditioned avoidance reflex in rats was fully preserved after 30 mg/kg sc (single dose) or 50 mg/kg po (for 4 consecutive days). After 30 mg/kg imipramine ip, mice maintained their body temperature; barbiturates were only slightly potentiated, and alcohol not at all. Tests on cats were also performed.

1248. Signorelli, S.

TOLERANCE FOR ALCOHOL IN PATIENTS ON CHLORPROPAMIDE.

Ann. N.Y. Acad. Sci. (New York), 74: 900-903 (0 ref.),

1959.

E – general – exp. comp. – DC (sensit.) – humans – acute admin. – chronic admin. – in vivo – blood lev. – blood comp., sites, lymph – cardiovasc. – CNS – G.I. tract – glands – hormones, hormone antag.

– *CAAAL-9024-D1

A-1318.

Chlorpropamide, a hypoglycemic agent which sometimes is superior to carbutamide or tolbutamide, particularly with maximum therapeutic doses, was effective in 72(80%) of 88 patients treated for diabetes mellitus for a minimum of 1 month. No toxic effects appeared, and the only 2 side effects were episodes of sensitivity and individual intolerance similar to those caused by other sulfonylurea derivatives, and episodes of specific gastrointestinal tract intolerance, namely: nausea, abdominal fullness, and constipation. 3-4 days after starting treatment, 8 of 23 patients experienced warmth, flushing, nausea, giddiness, or tachycardia after drinking wine with their meals. These symptoms disappeared quickly when no wine was taken. 1 such patient was switched to another sulfonylurea derivative, carbutamide, and experienced similar symptoms, which were mild and lasted only a few days. 8% ethanol administered to all alcohol-intolerant patients caused facial warmth and flushing within 20 min, and slight giddiness similar to that found after consumption of large quantities of alcohol. Normal subjects or diabetics being treated with other sulfonylurea derivatives experienced none of these symptoms after consuming the same alcohol dose. The affected patients had various parameters measured before and during the reaction: no electroencephalograph or significant pulse changes occurred; systolic blood pressure decreased about 20mm Hg, and diastolic, 5 mm Hg; and relevant auricular photoplethysmographic wave changes were noted.

1249. Simandl, J., and Franc, J.

ISOLACE TETRAETHYLTHIURAMDISULFIDU Z HNÍKU INKOUSTOVÉHO

(COPRINUS ATRAMENTARIUS). [The isolation of tetraethylthiuramdisulfide from the inky cap (*Coprinus atramentarius*)].

Chemicke Listy (Prague), 50: 1862-1863 (5 ref.),

1956.

C – exp. – DC (sensit.) – unclass. ther. agents – *CAAAL-0

A-1354.

The intolerance reaction to alcohol experienced after ingestion of *Coprinus atramentarius* is compared to that produced by disulfiram and cyanamide. Suspecting that this property of *Coprinus* is in fact due to the presence of disulfiram, the authors analyzed an autolytic sol of the fungus. A carbon tetrachloride extract was made under low temperature, and, when tested, it produced the characteristic brown colour of disulfiram. The solvent was evaporated, and methanol used to extract any further remaining fats. Upon evaporation of the methanol, light yellow needle-shaped crystals formed, with a melting point of 70.5°C—the same melting point as that of disulfiram. Due to the small quantity of test substance, a quantitative analysis was not possible. However, a paper chromatographic test was made on the substance and on disulfiram and tetramethylthiuram disulfide (TMTD), and the R_F values found for these compounds were, in the above order: 0.78, 0.78, 0.86. It is concluded that *Coprinus atramentarius* does contain disulfiram.

1250. Simandl, J., and Franc, J.

DIE ISOLIERUNG DES TETRAÄTHYLTHIURAMDISULPHIDS AUS DEM

TINTENMISTPILZ (*COPRINUS ATRAMENTARIUS*). [The isolation of tetraethylthiuramdisulfide from the inky cap (*Coprinus atramentarius*)].

Collection of Czechoslovak Chemical Communications (Prague), 22: 331-332 (5 ref.),

1957.

G – exp. – DC (sensit.) – unclass. ther. agents – *CAAAL-0

A-1355.

The intolerance reaction to alcohol experienced after ingestion of *Coprinus atramentarius* is compared to that produced by disulfiram and cyanamide. Suspecting that this property of *Coprinus* is in fact due to the presence of disulfiram, the authors analyzed an autolytic sol of the fungus. A carbon

tetrachloride extract was made under low temperature, and, when tested, it produced the characteristic brown colour of disulfiram. The solvent was evaporated, and methanol used to extract any further remaining fats. Upon evaporation of the methanol, light yellow needle-shaped crystals formed, with a melting point of 70.5°C—the same melting point as that of disulfiram. Due to the small quantity of test substance, a quantitative analysis was not possible. However, a paper chromatographic test was made on the substance and on disulfiram and tetramethylthiuram disulfide (TMTD), and the R_F values found for these compounds were, in the above order: 0.78, 0.78, and 0.86. It is concluded that *Coprinus atramentarius* does contain disulfiram.

1251. Simon, W., and Lucero, R. J.

CONSUMPTION OF MENTHOLATED CIGARETTES BY ALCOHOLICS.

Dis. Nerv. Syst. (Galveston), 21: 213-214 (0 ref.), 1960.
 E – stat. surv. – DC (add., infra-add., unspec. incr.) – humans – psychot. humans – drug-dep. humans
 – CNS – integ. syst. agents – *CAAAL-9247-Z1 A-1114.

The extent of consumption of mentholated cigarettes was studied in 4 different groups: (1) the normal population, (2) drug-dependent persons, (3) mental hospital patients, and (4) alcoholics. It was found that mental hospital patients in general, drug-dependent individuals, and the normal population smoke about the same proportion of mentholated cigarettes. Alcoholics, on the other hand, consume more mentholated cigarettes than any other group. It is hypothesized that, since menthol is a CNS stimulant, and alcohol is also a temporary stimulant, the 2 agents may well have a synergistic action, producing an augmented effect.

1252. Simsch, A.

TIEREXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE GEMEINSAME WIRKUNG VON ALKOHOL UND D-CYCLOSERIN. [Experimental investigations on the combined effects of alcohol and D-cycloserine on animals].

Dissertation, Faculty of Medicine of the Free University of Berlin, West Germany, 75 pp. (49 ref.), 1965.

G – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – anti-infectants – *CAAAL-0 B-1021.

To investigate the combined effects of cycloserine (CS) and 96% ethanol, the LD_{50} of CS was determined in white mice, and was found to be 11,980 mg/kg. 214 mice (19 groups of 8-20 animals) were then given decreasing fractions of the LD_{50} of each substance, combined with a constant dose of the other substance. Time to onset of narcosis, duration of narcosis, and survival time were recorded, and observations of toxic effects were made for 12-24 hr. Small CS doses considerably increased alcohol toxicity, corresponding to the LD_{50} , whereas small alcohol doses substantially decreased the expected LD_{50} CS toxicity. Toxic phenomena—ataxia, clonic spasms of all extremities, diarrhea, apathy, eye damage, and a Parkinsonian-like shaking of the extremities—were more severe in animals given the combination. Time to onset of narcosis, duration of narcosis, and survival time were all significantly shorter for the combined dose.

1253. Singh, J. M.

EFFECT OF ACUTE AND CHRONIC ADMINISTRATION OF ETHYL ALCOHOL ON THE DEVELOPMENT OF TOLERANCE TO PENTOBARBITAL.

Industr. Med. Surg. (Chicago), 39(7): 306 (0 ref.), 1970.

E – abst. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – CNS – barbiturates – *CAAAL-0 B-0989.

Previous findings of the author have established that developed tolerance to pentobarbital and thiopental is characterized by a decrease in sleeping time or hypnotic effect, and that the state of the

thyroid gland is implicated in the development of this tolerance. In the present reported experiments, rats were divided into 4 groups: the first group received 30 mg pentobarbital (P); the second, P (same dose) + 2.5% ethanol (E); the third, P + 10% E; and the fourth, P + 15% E. Ethanol was added to the drinking water for 32 days. After determining the initial Percentage Tolerance Index (PTI = 100 x ratio of hypnotic effect of the first injection/hypnotic effect of the second injection), sleeping time was determined on days 15 and 31. On days 16 and 32, pentobarbital was injected, and, after loss of righting reflex, 20 ml of 2.5%, 10.0%, or 15% E was administered. In acute administration of E, developed tolerance to pentobarbital was reversed; however, with chronically-treated rats, developed tolerance to pentobarbital was not reversed. It is suggested that acute ethanol administration can reverse developed tolerance to pentobarbital, whereas chronic ethanol feeding can enhance it.

1254. Singh, J. M., Trahan, P., and Liljeberg, J. A.

THE REVERSAL OF DEVELOPED TOLERANCE TO PENTOBARBITAL BY ETHYL ALCOHOL.

Arch. Int. Pharmacodyn. (Gand), 184(2): 334-342 (13 ref.), 1970.
 E – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – liver, kidney – metab. proc. – barbiturates – *CAAAL-0 B-0498.

The effect of ethyl alcohol (EA) on developed tolerance to pentobarbital (Pb) was studied in rats. 30 mg/kg Pb was administered ip to the rats on days 1 and 2, and again on days 8 and 9 of the experiment. The difference in sleeping times after drug injection gave a measurement of the tolerance developed. Significant tolerance to Pb was developed on the second day in all cases. 1.495 and 2.243 EA, administered on the ninth day, reversed the developed tolerance, whereas 0.373 g/kg EA partially reversed the tolerance. Administration of 1.495 g/kg EA on the ninth day, 30 and 60 min before Pb, also reversed developed tolerance, as did 1.495 and 2.243 g/kg EA administered on the eighth and ninth days after Pb. It is concluded that certain doses of EA are able to reverse developed tolerance to Pb. The decrease in rectal body temperature during the development of tolerance suggests the involvement of the CNS in this process.

1255. Singh, J. M.

COMPARISON BETWEEN ACUTE AND CHRONIC ADMINISTRATION OF ETHYL ALCOHOL ON THE DEVELOPMENT OF TOLERANCE TO PENTOBARBITAL.

Arch. Int. Pharmacodyn. (Gand), 189(1): 123-128 (9 ref.), 1971.
 E – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – dose resp. – CNS – liver, kidney – metab. proc. – barbiturates – *CAAAL-0 B-0990.

Pentobarbital was given to non-starved rats in 8 iv injections of 30 mg/kg over a 49-day period, and the effects of acute and chronic ethanol administration on pentobarbital tolerance, measured on the basis of changes in sleeping time, were determined. In acute experiments, 20 ml/kg of 2.5, 10.0, or 15.0% ethanol was administered rectally on the 17th day. In chronic experiments, 2.5, 10.0, or 15% ethanol was added to the drinking water up to the 49th day. Sleeping time was determined on the 32nd, 33rd, 48th, and 49th days. Significant tolerance to pentobarbital was developed on the 1st, 17th, 33rd, and 49th day of chronic ethanol administration. Tolerance to pentobarbital developed within 24 hr. It was reversed by the acute rectal administration of ethanol; however, a dose which reversed the tolerance in these rats not only did not reverse, but enhanced the process of tolerance in chronically ethanol-treated rats. Rectal temperature decreased during the development of pentobarbital tolerance and during chronic ethanol administration. Explanations for the opposite effects of acute and chronic ethanol administration are discussed on the basis of metabolism and diuresis.

1256. Sinitsyn, S. N.

IZMENENIE AKTIVNOSTI KHOLINESTERAZY POD VLIANIEM TSIANIDOV I ALKOGOLIA PRI IKH RAZDEL'NOM I KOMBINIROVANNOM DEISTVII. [Changes in cholinesterase activity under the influence of cyanides and alcohol acting separately and in combination].

Farmakol. Toksik. (Moscow), 24: 540-541 (0 ref.),

1961.

R – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – unclass. ther. agents – *CAAAL-10022-B2 A-1115.

The cholinesterase activity in 114 rats and 206 mice was studied under the following experimental conditions: (1) control, (2) after 2-4 mg/kg potassium cyanide, (3) after 0.0075 ml 40% alcohol/g, (4) after alcohol, followed 20 min later by cyanide administration, and (5) after cyanide, followed 10 min later by alcohol. Group (5) was decapitated 10 min after the alcohol, and groups (3) and (4) 20 min after alcohol. Serum cholinesterase activity (SCA) almost doubled 8-10 min after cyanide, and decreased by about 13% after alcohol. Alcohol given prior to cyanide counteracted the effect of the latter, and prevented an increase of SCA; when given subsequent to cyanide, SCA increase after cyanide was not diminished, but prolonged.

1257. Sinitsyn, S. N.

IZMENENIE SAKHARA KROVI POD VLIANIEM TSIANIDOV I ALKOGOLIA PRI IKH RAZDEL'NOM I KOMBINIROVANNOM DEISTVII. [Blood sugar changes under the influence of cyanides and alcohol in their separate and combined use].

Farmakol. Toksik. (Moscow), 25: 113-114 (0 ref.),

1962.

R – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – liver, kidney – unclass. ther. agents – *CAAAL-10034-B2 A-1116.

Changes in blood sugar were studied in 126 white rats. 10 min after 3 mg/kg potassium cyanide po, the amount of blood sugar sharply increased to an average of 155.5 mg% (compared with an average of 113 mg% in controls). 3 hr after 0.0075 ml/g 40% alcohol po, there was a tendency towards a decreased blood sugar content. Alcohol given prior to cyanide (3 mg/kg) inhibited the increase in blood sugar (average blood sugar concentration of 134 mg%, compared to 155.5 mg% for cyanide alone). Alcohol administered after cyanide increased the blood sugar to an average of 157.5 mg%.

1258. Sirnes, T. B.

DRUGS AND DRIVING.

In: *Alkohol und Verkehrssicherheit: Konferenzbericht der 5. Internationalen Konferenz über Alkohol und Verkehrssicherheit*. [Alcohol and Traffic Safety: proceedings of the 5th International Conference on Alcohol and Traffic Safety]. Freiburg im Breisgau, West Germany, 1969. Freiburg im Breisgau: Hans Ferdinand Schulz Verlag, pp. III.39-III.43 (0 ref.),

1969.

E – SEC – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – drug-dep. humans – blood lev. – CNS – barbiturates – tranquilizers – *CAAAL-0 B-0582.

The author discusses the legal and pharmacological aspects involved in 21 Norwegian lawsuit cases from 1963-1966, concerning drivers operating a motor vehicle while intoxicated by a drug or a combination of a drug and alcohol. Clinical proof of intoxication was available for 18 drivers. 9 cases involved alcohol-drug combinations (blood alcohol levels ranged from 0.27-0.023%); 11 cases involved barbiturates, alone or together with psychopharmacological agents or stimulant drugs; 5 cases involved meprobamate; 3 cases amitriptyline (tryptizol or saroten); and only 3 cases involved the newer anti-anxiety drugs, chlordiazepoxide (librium) or diazepam (valium). Certainly there is also a large group of drivers who take psychoactive drugs in doses which do not produce obvious symptoms and which cannot be readily detected clinically, but which nevertheless reduce driving ability.

1259. Sjövall, H., and Voigt, G.

EINIGES ÜBER DAS ERSCHEINUNGSBILD DES CHRONISCHEN ALKOHOLISMUS.

[Some aspects of the picture of chronic alcoholism].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 46: 30-41 (7 ref.),

1957.

G – stat. surv. – DC (add., infra-add., unspec. incr.) – DC (sensit.) – post-mort. – drug-dep. humans – blood lev. – sed., hypnot. – *CAAAL-8521-D3

A-1117.

Observations from 145 autopsies on Swedish alcoholics made at the University of Lund Institute of Forensic Medicine are reported. The pathological changes in Swedish alcoholics differ in many respects from the rest of Scandinavia and Central Europe. Suicides were common. The significance of alcohol-drug interactions is discussed. The lower tolerance of alcoholics to barbiturates and morphine, and the additive effect of disulfiram are well known. 5 deaths occurred from combined use of alcohol and promethazine. The case of a 55 yr-old man who took an undetermined amount of promethazine after alcohol ingestion is described; he died shortly afterwards with a blood alcohol concentration of 2.0°/oo. The authors conclude that the use of promethazine in treating alcoholics should be discontinued.

1260. Small, M. D., Gershoff, S. N., Broitman, S. A., Colon, P. L., Cavanagh, R. C., and Zamcheck, N. EFFECT OF ALCOHOL AND DIETARY DEPRIVATION ON ABSORPTION OF XYLOSE FROM THE RAT SMALL INTESTINE.

Amer. J. Dig. Dis. (New York), 5(9): 801-806 (8 ref.),

1960.

E – exp. cont. – congen. stud. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. – in vivo – absorp., distrib., stor. – G.I. tract – diagnost. agents – miscellaneous – *CAAAL-9814-B2

A-1118.

In studies on rats on either a complete or protein deficient diet, it was found that those drinking alcohol absorbed much less xylose than those which did not. In one study, 20 rats received for 1 yr a complete diet plus: water, a 20% alcohol sol, water with congeners of distilled beverages, or the alcohol sol with the congeners. The rats drinking the alcohol with congeners absorbed the least xylose (74 mg), and those receiving water with congeners the most (132 mg). Rats drinking water and alcohol absorbed 112 and 96 mg, respectively. The mechanism whereby alcohol diminishes xylose absorption is not clear.

1261. Smart, R. G., Schmidt, W., and Bateman, K.

PSYCHOACTIVE DRUGS AND TRAFFIC ACCIDENTS: A STUDY OF PERSONS DEPENDENT ON STIMULANTS, DEPRESSANTS, TRANQUILLIZERS AND ALCOHOL.

In: *Alkohol und Verkehrssicherheit: Konferenzbericht der 5. Internationalen Konferenz über Alkohol und Verkehrssicherheit*. [Alcohol and Traffic Safety: proceedings of the 5th International Conference on Alcohol and Traffic Safety]. Freiburg im Breisgau, West Germany, 1969. Freiburg im Breisgau: Hans Ferdinand Schulz Verlag, pp. III.45-III.51 (7 ref.),

1969.

E – SEC – stat. surv. – conj. addict. – mot. vehic. – drug-dep. humans – CNS – amphetamines – barbiturates – tranquilizers – *CAAAL-0

B-0560.

The incidence of automobile accidents among abusers of psychoactive drugs was investigated. Of the 30 drug abusers studied, 15 were "mixed addicts" who, besides being alcoholics, were also dependent upon some psychoactive drug. The drug users were questioned about their driving experiences, both after drug and alcohol ingestion, and while sober for the yr 1961-1966. It was found that substantially more drug abusers were involved in accidents than was the general population, the rate being about 1.9 times higher. The 7 addicts who used alcohol and barbiturates had only 2 observed drivers in accidents, compared to an expectancy of 2.584. The 5 who used alcohol and tranquilizers, with or without barbiturates, had 3 times as many drivers involved in accidents than expected (observed = 5, expected = 1.610). It is concluded that drug addicts who combine use of alcohol and barbiturates

have fewer accidents than expected for their age, sex, and exposure in terms of miles driven, whereas those dependent on alcohol and tranquilizers have significantly elevated rates.

1262. Smart, R. G., Schmidt, W., and Bateman, K.
PSYCHOACTIVE DRUGS AND TRAFFIC ACCIDENTS.
 Journal of Safety Research (Chicago), 1(2): 67-73 (24 ref.), 1969.
 E – SEC – stat. surv. – conj. addict. – mot. vehic. – drug-dep. humans – CNS – amphetamines –
 barbiturates – tranquilizers – *CAAAL-0 B-0451.

The accident rates of 30 psychoactive drug abusers were studied. The group included persons dependent on barbiturates, tranquilizers, and stimulants, and, in addition, 1/2 of the group were also dependent on alcohol. The rate of accidents and driving exposure (miles driven between 1961 and 1966) were compiled; expected accident rates/10,000 miles were computed and compared with the observed rates. It was found that the group as a whole had double the expected accident rate, the major part of the excess being due to the subjects dependent on amphetamine (alone or in combination). Those dependent on alcohol plus barbiturates, barbiturates only, or tranquilizers only had lower than expected rates. The 5 alcohol plus barbiturate-dependent subjects had an observed rate of 2 accidents/10,000 miles, compared to an expected rate of 2.584; the 3 alcohol plus tranquilizer-dependent subjects had an observed rate of 5 and an expected rate of .914; the 2 persons addicted to alcohol plus tranquilizers and barbiturates had an observed rate of 0, compared to .696; and the observed rate for the 3 subjects addicted to amphetamines plus alcohol and barbiturates or tranquilizers was 1, compared to 1.258.

1263. Smetana, H.
NEPHROSIS DUE TO CARBON TETRACHLORIDE.
 Arch. Intern. Med. (Chicago), 63: 760-777 (51 ref.), 1939.
 E – SEC – general – case hist. – DC (add., infra-add., unspec. incr.) – drug-dep. humans – other drug
 lev. – absorp., distrib., stor. – cardiovasc. – CNS – G.I. tract – liver, kidney – respir. – anti-infectants
 – *CAAAL-0 A-1119.

3 cases of carbon tetrachloride poisoning are presented, 2 of which were fatal. In all 3 cases, there was a history of chronic alcoholism, or of steady and heavy drinking. In 1 of the cases, the co-worker of the patient, a teetotaler, had been working in the same room, and had been exposed to the vapours of carbon tetrachloride for the same length of time, yet, he was relatively unaffected, whereas the patient in question died.

1264. Smilga, J.
**ABSCHWÄCHUNG UND AUFHEBUNG DER LOKALANÄSTHESIERENDEN
 WIRKUNG DES KOKAINS DURCH GEWÖHNUNG AN ALKOHOL.** [Decrease and
 cancellation of the local anesthetic-effect of cocaine through habituation to alcohol].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 171:
 162-169 (15 ref.), 1933.
 G – exp. cont. – DC (decrease) – mammals – acute admin. – chronic admin. – in vivo – metab. proc.
 – skel., muscle, skin – anesthetics – *CAAAL-0 A-1120.

In tests on the cornea of guinea pigs, alcohol (8 cc/kg 25% sol/day for 4 weeks) weakened and neutralized the anesthetic effect of cocaine (2 drops 0.75% sol/kg) after a few days. No weakened effect was observed in animals not habituated to alcohol. It is concluded that alcohol is a poison which has a static as well as a dynamic action. The more the static effect increases with habituation, the more it pathobiotically transforms the normal state of the tissue. A desensitizing inversion of the dynamic effect, which weakens the cocaine anesthesia, results (pathobiotic desensitization). Conjectures are made on the reaction mechanism. The effect is likened to morphine habituation leading to

chronic poisoning. The pathobiotic effect of alcohol goes on for weeks after administration is terminated.

1265. Smillie, W. G., and Pessoa, S. B.

TREATMENT OF HOOKWORM DISEASE WITH CARBON TETRACHLORIDE.

American Journal of Hygiene (Baltimore), 3: 35-45 (12 ref.),

1923.

E – SEC – general – DC (add., infra-add., unspec. incr.) – drug-dep. humans – other drug lev. – G.I. tract – liver, kidney – anti-infectants – *CAAAL-0

A-1121.

Experiments were performed on dogs and humans. In human subjects, it was found that 1 cc and 3 cc of carbon tetrachloride (CCl_4) were remarkably effective in treating hookworm disease (95-97% efficiency); 3 cases of intoxication subsequently appeared—all were alcoholics. Thus, the recommended dosage of 3 cc for adults should not be followed in those cases in which the liver is already damaged. CCl_4 is dangerous to administer directly, following an alcoholic debauch or to chronic alcoholics, as even so small a dose as 1.5 cc can produce severe intoxication.

1266. Smith, A. A., Karmin, M., and Gavitt, J.

BLOCKING EFFECT OF PUROMYCIN, ETHANOL, AND CHLOROFORM ON THE DEVELOPMENT OF TOLERANCE TO AN OPIATE.

Biochem. Pharmacol. (New York), 15: 1877-1879 (8 ref.),

1966.

E – exp. cont. – exp. comp. – cross-tol. – mammals – acute admin. – in vivo – CNS – senses – analg., antipyret. – *CAAAL-0

B-0452.

Because of the presumed central origin of tolerance to the lenticular effects of levorphanol, it was speculated that substances with anesthetic properties might influence the development of tolerance. In female mice, ethanol (25% sol in normal saline (v/v)) in a dose of 4 g/kg ip virtually prevented the development of tolerance to levorphanol (75 μ moles/kg sc). Chloroform was also effective. The administration (24 hr previously) of ethanol without levorphanol did not affect the subsequent response to an ED_{80} of levorphanol.

1267. Smith, A. A., and Hayashida, K.

BLOCKADE BY PROPRANOLOL OF THE RESPIRATORY AND NARCOTIC EFFECTS OF ETHANOL.

In: *Committee on Problems of Drug Dependence, 1970. Report of the Thirty-Second Meeting, February 16, 17, and 18, 1970. Washington: National Academy of Sciences-National Research Council,*

pp. 6879-6882 (6 ref.) @ 1970.

E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – acid-base, blood pH, elect. – CNS – respir. – stimulants – *CAAAL-0

B-0991.

Female Swiss-Webster mice (20-25 g) received no drug, 1, 2, or 3 g/kg ethanol, or 3 g/kg ethanol plus 1 of the following: 1 mg/kg epinephrine, 1 mg/kg isoproterenol, 10 mg/kg phenoxybenzamine, 1 mg/kg propranolol, or 5 mg/kg *d*-amphetamine. Ethanol was injected ip as a 25% (w/v) saline sol; the other drugs were injected ip in a vol of 0.2-0.3 ml, 10 min before the ethanol. The changes in capillary blood pH and pCO_2 were then determined. As the dosage increased, ethanol caused a linear fall in blood pH, and a concomitant rise in pCO_2 . The amines intensified ethanol-induced respiratory depression, whereas the β -blocker, propranolol, completely inhibited the ethanol effect. The α -blocker, phenoxybenzamine, had no significant effect on respiration. When propranolol (1 mg/kg) was given 15 min prior to 16 mg/kg morphine or 40 mg/kg meperidine, it failed to counteract opioid-induced respiratory depression. A study of the effect of propranolol pretreatment on the time of return of righting reflexes (TRRR) after injection of 4 g/kg ethanol or 60 mg/kg pentobarbital revealed a TRRR of nearly 40 min in mice given the ethanol alone, in contrast to a far shorter TRRR

in animals treated 15 min previously with 105 mg/kg propranolol. Higher doses of propranolol, however, potentiated ethanal narcosis. Small doses of propranolol did not block barbiturate narcosis, indicating different mechanisms of narcosis for ethanol and pentobarbital.

1268. Smith, C. M.

A NEW ADJUNCT TO THE TREATMENT OF ALCOHOLISM: THE HALLUCINOGENIC DRUGS.

Quart. J. Stud. Alcohol (New Haven), 19: 406-417 (16 ref.), 1958.
E – SEC – exp. comp. – cross-tol. – drug-dep. humans – acute admin. – in vivo – hallucinogens – *CAAAL-8293-M3 A-1444.

The effectiveness of single doses of LSD (200-400 μ g po) and mescaline (0.5 g po) in treatment of alcoholics was evaluated. A psychotherapeutic relationship was gradually established (2-4 weeks), following which 1 of the hallucinogens was administered, and a prolonged interview conducted, during which problems were discussed and negative suggestions made regarding alcohol use. A follow-up investigation revealed 6 patients much improved, 6 improved, and 12 unchanged, and it is concluded that further trials are justified. It was also found that the alcoholics were remarkably resistant to the drugs. Whereas, in normal subjects, 100 μ g LSD is sufficient to provoke a profound reaction in 80% of all cases, and 200 μ g is sufficient in 100% of all cases, 200-400 μ g were needed to produce comparable results in the observed group. Mescaline, on the whole, produced the more marked reactions.

1269. Smith, H. W.

PHARMACOLOGY OF ALCOHOL AND ALCOHOL-DRUG COMBINATIONS.

In: Harger, Rolla N., ed. *Alcohol and traffic safety*. Proceedings of the Fourth International Conference on Alcohol and Traffic Safety at Indiana University, December 6-10, 1965. Bloomington, Indiana: Indiana University Press, pp. 26-32 (23 ref.), 1966.
E – SEC – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – CNS – amphetamines – barbiturates – *CAAAL-0 B-0453.

Literature on the topic, including a brief mention of alcohol-drug interactions, is reviewed. The present inadequacy of statistical documentation on the problem of drugs and driving is stressed. More research is needed on topics like driving behaviour, alcohol and drug effects, drug combinations as they affect driving, and the types of people who are driving after alcohol and drug ingestion, in order to reappraise our approaches in education, enforcement, and treatment.

1270. Smith, J. W., and Loomis, T. A.

THE POTENTIATING EFFECT OF ALCOHOL ON THIOPENTAL INDUCED SLEEP.

Proc. Soc. Exp. Biol. Med. (New York), 78: 827-829 (6 ref.), 1951.
E – exp. cont. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – blood lev. – mot. perform. – CNS – sed., hypnot. – *CAAAL-6105-D2 A-1122.

2 experiments were performed. The effect of 1000 mg/kg alcohol iv, 5 mg/kg thiopental iv, or both substances in combination, on the reaction time of mice to a painful stimulus was tested. Secondly, sleeping time was recorded in mice, rabbits, and dogs after alcohol iv (1000 mg/kg 10% alcohol sol in isotonic saline), thiopental iv (mice—30 mg/kg, rabbits—25 mg/kg, and dogs—20 mg/kg), or both substances in combination with the above dosages. In the first experiment, the mice reacted more quickly after thiopental alone, but, after alcohol alone or alcohol-thiopental, reaction times were not significantly affected. In the second experiment, sleeping time was significantly increased after alcohol-thiopental, as compared to either substance administered alone; the combined effect was greater than the sum of the individual effects.

1271. Smith, M. E., and Newman, H. W.

THE RATE OF ETHANOL METABOLISM IN FED AND FASTING ANIMALS.

J. Biol. Chem. (Baltimore), 234: 1544-1549 (24 ref.),

1959.

E – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – in vitro – blood lev. – species or sex diff. – liver, kidney – metab. proc. – anti-infectants – miscellaneous – *CAAAL-8818-A2

A-1123.

The rate of alcohol oxidation was determined in rat liver slices. Slices from fasted rats metabolized alcohol at 1/2 the rate of slices from fed rats (0.33 mg/100 mg and 0.66 mg/100 mg, respectively) in a 3 hr period. 5 mg diphosphopyridine nucleotide (DPN) increased the oxidation rate in slices from fasted rats to 0.42 mg/100 mg, but failed to affect the oxidation rate in slices from fed rats. Semicarbazide, acetaldehyde, and acetate, added to the incubation medium, failed to affect the disappearance of alcohol from slices of fed or fasted animals. Methylene blue, ferricyanide, and sodium pyruvate were added to the incubation medium. Methylene blue and ferricyanide raised the rate in slices from fasted animals, but not in those from fed animals, whereas pyruvate increased alcohol metabolism in livers from both fed and fasted animals. Pyruvate and alanine both increased the DPN: DPNH (reduced form) ratio and the rate of alcohol metabolism. In vivo studies in dogs showed that, after 3 g/kg 15% alcohol sol iv, with or without alanine or pyruvate by stomach tube, pyruvate did not affect the rate of alcohol metabolism, whereas alanine (4.9% concentration) increased the rate in all animals.

1272. Smith, M. E.

INTERRELATIONS IN ETHANOL AND METHANOL METABOLISM.

J. Pharmacol. Exp. Ther. (Baltimore), 134: 233-237 (15 ref.),

1961.

E – exp. cont. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – in vitro – blood lev. – species or sex diff. – liver, kidney – metab. proc. – enzymes – *CAAAL-10603-A2

A-1124.

In vivo and in vitro studies were conducted on rats. The blood ethanol disappearance curves were plotted for rats given 1, 2, or 4 g ethanol/kg, with and without pretreatment with 3-amino-1,2,4-triazole (AT); there was no difference found between the normal and catalase-inhibited animals. Liver slices from normal and AT-treated rats were incubated with equimolar amounts of ethanol or methanol added to the medium; no difference was observed in the amount of ethanol metabolized in the normal and AT-treated liver slices, while the oxidation of methanol by AT-treated liver was 30% of the normal rate. The oxidation of ethanol and methanol was studied in the presence of xanthine; the rate of methanol oxidation was more than doubled, whereas that of ethanol was unaffected. In equimolar amounts, ethanol inhibited methanol metabolism in both the rat liver slices and a pure beef catalase system by about 1/2. Methanol did not inhibit ethanol oxidation in the liver slices, but, in the pure beef catalase system, some inhibition was seen.

1273. Smith, M. E., Evans, R. L., Newman, E. J., and Newman, H. W.

PSYCHOTHERAPEUTIC AGENTS AND ETHYL ALCOHOL.

Quart. J. Stud. Alcohol (New Haven), 22(2): 241-249 (5 ref.),

1961.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – in vitro – dose resp. – blood lev. – CNS – liver, kidney – metab. proc. – respir. – antidepressants – enzymes – tranquilizers – *CAAAL-9675-A2

A-1125.

6 female dogs were exposed to the following experimental conditions: 1 or 2 mg/kg chlorpromazine im, followed 1/2 hr later by 3 g/kg ethanol as a 15% sol (w/v) iv, which was given over a 30 min period; 10 mg/kg iproniazid po, given daily for 2 days before, and also 1/2 hr before, the above alcohol dose; and 1/2, 1, and 3 mg/kg pheniprazine po, given daily for 2 days before, and also 1/2 hr before, the above alcohol dose. Rats received 2.5 mg chlorpromazine/rat/day for 2 days before, and also 1/2 hr before, being sacrificed and the liver slices inserted into flasks containing 10.0 mg ethanol in 4.0

ml of 0.1 M phosphate buffer; in another in vitro test, the same procedure was followed with pheniprazine (1 mg pheniprazine/rat/day for 5 days, or 1 or 2 mg 1 hr before sacrifice). An in vivo test with chlorpromazine-alcohol was also conducted on rats. The results showed that all 3 drugs increased the depression induced by alcohol in the dog. Also, high doses of chlorpromazine and pheniprazine slowed the rate of alcohol metabolism. In vitro studies with rat liver slices, with pheniprazine in the incubation medium, also showed an inhibition of alcohol metabolism, whereas chlorpromazine and iproniazid were without effect.

1274. Smith, S. E., and Herxheimer, A.

TOXICITY OF ETHANOL-BARBITURATE MIXTURES.

J. Pharm. Pharmacol. (London), 21: 869-870 (3 ref.),

1969.

E – general – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mammals – CNS – barbiturates – *CAAAL-0 B-0499.

In a letter to the editor, the authors disagree with the view of Wiberg, Coldwell, and Trenholm (J. Pharm. Pharmacol., 21(4): 232-236, 1969) that ethanol and barbiturates supra-additively interact. Instead, the data from this study, concerning acute toxicity of ethanol-barbiturate mixtures in male rats, calculated from mortality figures, should be interpreted differently by the plotting of isobols (“lines on a combined dose diagram connecting those dose pairs which are equi-effective in producing a defined pharmacological effect”). No more than a single additive effect was indicated by the line in each case. In addition, the data on the prolongation of sleeping time was insufficient for isobol construction, but the fact that 2 inactive doses, when combined, produced a marked effect, can be explained by summation just as easily as by potentiation or supra-addition. In an attempt to distinguish these 2 possibilities, it is recommended that combinations of half the original doses, or, alternatively, the effects of double the original doses of either drug alone, be tested.

1275. Smyth, H. F., Smyth, H. F., Jr., and Carpenter, C. P.

THE CHRONIC TOXICITY OF CARBON TETRACHLORIDE; ANIMAL EXPOSURES AND FIELD STUDIES.

Journal of Industrial Hygiene and Toxicology (Cambridge, Mass.), 18(5): 277-298 (23 ref.),

1936.

E – SEC – general – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – liver, kidney – anti-infectants – *CAAAL-0 A-1126.

On the basis of animal experiments with guinea pigs, monkeys, and rats, it is concluded that an average of 100 parts per million of carbon tetrachloride vapour is a safe concentration for continuous exposure of workmen throughout the day, and for prolonged periods. Of 96 men exposed to carbon tetrachloride in industry, tests showed none who could be considered seriously or unmistakably injured by the solvent vapours. The authors note, however, that the use of alcohol, as well as dietary factors, can enhance one's susceptibility to the toxic effects of this chemical. The degree of cirrhosis found in 1 chronic alcoholic was considered to have been undoubtedly increased by carbon tetrachloride exposure.

1276. Söderström, N.

NÅGRA ORD OM METHANOLFÖRGIFTNING. [Some remarks on methanol poisoning].

Svenska Läkartidningen (Stockholm), 44(17): 960-967 (4 ref.),

1947.

S – general – case hist. – DC (antidotal) – humans – blood lev. – metab. proc. – alcohols – analg., antipyret. – autonomic agents – *CAAAL-0 A-1127.

The author discusses poisoning effects by methanol-adulterated alcohol, and gives specific case histories. On the basis of his experience, the author feels that the value of ethanol treatment is prophylactic only in the early stages of methanol intoxication under hospitalization conditions. It is

pointed out that a constant blood alcohol concentration of 0.1% is adequate to inhibit the toxic effects of methanol by arresting its decomposition. The initial dose is 50-75 ml pure ethanol, and then approximately 10 ml/hr, to maintain satisfactory blood alcohol concentrations. 15 cl alcohol administered to a patient suffering from advanced methanol poisoning, who had earlier been sedated with morphine-scopolamine, could not save the patient from exitus two hr later.

1277. Soehring, K.

VERSTÄRKUNG VON BARBITURSÄUREWIRKUNGEN DURCH ANDERE ARZNEIMITTEL. [Reinforcement of the effects of barbituric acid by other drugs].

Med. Klin. (Munich), 48(15): 537-539 (22 ref.),

1953.

G – SEC – review – DC (add., infra-add., unspec. incr.) – mammals – CNS – miscellaneous – sed., hypnot. – *CAAAL-0 A-1128.

A number of papers are reviewed which report investigations on the effects of glucose, alcohol, disulfiram, myanesin, curare, iodine mediations, cholesterine, and other compounds on the duration and depth of barbiturate narcosis in animals. It is noted that alcohol prolongs the sleep induced by barbiturates, even if it is inhaled as a vapour.

1278. Soehring, K., and Schmidt, G.

DIÄTHYLBARBITURSÄURE. [Diethylbarbituric acid].

Munchen. Med. Wschr. (Munich), 104(41): 1939-1940 (8 ref.),

1962.

G – ES – general – DC (add., infra-add., unspec. incr.) – humans – CNS – barbiturates – *CAAAL-0 A-1129.

The authors comment on the adverse properties of diethylbarbituric acid, pointing out that the combined use of diethylbarbituric acid and other drugs, including alcohol, may lead to synergism, with serious effects on driving ability. Of all the barbiturates, diethylbarbituric acid has the largest medical dose (0.5 g) and the longest duration of action (6-8 hr after a single dose and subthreshold after-effects for several days). Therefore, synergistic effects with alcohol can occur days after administration of the drug. In conclusion, the authors suggest that diethylbarbituric acid prescriptions should be issued with great caution. Its replacement by other barbiturate sedatives is recommended, in view of its high single dose and its long-lasting effects.

1279. Soehring, K.

ARZNEIMITTEL ALS UNFALLURSACHE IM STRASSENVERKEHR. [Drugs as a cause of accidents in traffic].

In: Möller, H., ed. *Versicherungsmedizin und Versicherungsrecht: Festschrift für Hans Göbbels*. Karlsruhe: Verlag Versicherungswirtschaft, pp. 167-170 (0 ref.),

1964.

G – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – med.-leg. – mot. vehic. – humans – mammals – mot. perform. – CNS – metab. proc. – analg., antipyret. – *CAAAL-0 A-1130.

The difficulties facing physician, judge, and lawyer in correctly assessing drug-alcohol interactions and driver impairment are discussed. 1 important case of the OLG Celle (see: Oberlandesgericht Celle. [Superior District (Appeal) Court, Celle]: *Neue Juristische Wochenschrift* (Berlin), 16: 2385-2386, 1963) decided that, if a defendant pleads that he was incapable of recognizing his inability to drive, due to the enhancement of the alcohol effect by a drug (butazolidin), an exceptionally thorough medical examination is warranted. What is significant is not the general alcohol-drug interaction, but the actual effect exhibited in the accused. A test to duplicate the condition has been possible only in a few cases, due to the necessary expense and possible dangers of the test. There is always the objection that the conditions on the day of the test differed from those of the accident, and an ideal medical examiner who has had a wide experience in the field of drug interactions is hard to find.

1280. Soehring, K., and Schüppel, R.
 WECHSELWIRKUNGEN ZWISCHEN ALKOHOL UND ARZNEIMITTELN. [Interactions between alcohol and drugs].
 Deutsch. Med. Wschr. (Stuttgart), 91(42): 1892-1896 (34 ref.), 1966.
 G – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals – blood lev. – species or sex diff. – absorp., distrib., stor. – CNS – metab. proc. – skel., muscle, skin – barbiturates – tranquilizers – *CAAAL-0 B-0454.

The subject of alcohol-drug interactions, and experimental evidence to date are reviewed. The importance of the need for study in this area is stressed. The various methods of study—evaluation of clinical data, psychological tests in humans, psychopharmacological studies in animals, and biochemical and pharmacodynamic studies in vitro and in vivo are discussed in detail. It is noted that surprisingly little clinical data from reliable observations have been compiled. Most observations are to be found in unpublished opinions, conclusions, and reports of the pharmaceutical industry; these indicate that interactions with alcohol are particularly likely with the following compounds: hypnotics or anti-epileptics, psychotropic drugs (including antidepressants), isoniazid and other monoamine oxidase inhibitors, antihistamines, oral antidiabetics, analgesics of the morphine type, and pyrazolones.

1281. Soehring, K., and Schüppel, R.
 INTERACTIONS BETWEEN ALCOHOL AND DRUGS.
 German Med. Monthly (Stuttgart), 12(2): 87-90 (34 ref.), 1967.
 E – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals – blood lev. – species or sex diff. – absorp., distrib., stor. – CNS – metab. proc. – skel., muscle, skin – barbiturates – tranquilizers – *CAAAL-0 B-0455.

The subject of alcohol-drug interactions, and experimental evidence to date are reviewed. The importance of the need for study in this area is stressed. The various methods of study—evaluation of clinical data, psychological tests in humans, psychopharmacological studies in animals, and biochemical and pharmacodynamic studies in vitro and in vivo are discussed in detail. It is noted that surprisingly little clinical data from reliable observations have been compiled. Most observations are to be found in unpublished opinions, conclusions, and reports of the pharmaceutical industry; these indicate that interactions with alcohol are particularly likely with the following compounds: hypnotics or anti-epileptics, psychotropic drugs (including antidepressants), isoniazid and other monoamine oxidase inhibitors, antihistamines, oral antidiabetics, analgesics of the morphine type, and pyrazolones.

1282. Soehring, K.
 EINSCHRÄNKUNG DER FAHRTÜCHTIGKEIT DURCH ALKOHOL UND ARZNEIMITTEL. [Impairment of driving ability by alcohol and drugs].
 Pharmazie (Berlin), 22(4): 222 (0 ref.), 1967.
 G – abst. – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – blood lev. – analg., antipyret. – barbiturates – *CAAAL-0 B-0456.

The steadily growing consumption of alcohol and drugs, and the increasing use of motor vehicles are discussed. While the determination of the blood alcohol level is a matter of routine, the detection of drugs in the body is still difficult. Hundreds of different drugs with different chemical compositions influence driving ability, and each one has a different metabolic fate in the body. Many of the drugs have synergistic effects if ingested simultaneously with alcohol, but no drug is known as yet which has a significant effect on the blood alcohol level.

1283. Soehring, K., and Wolters, H. G.
 PHARMAKOLOGISCHE GRUNDLAGEN DER WIRKUNG VON ARZNEIMITTELN AUF DIE VERKEHRSTÜCHTIGKEIT. [Pharmacological principles of the effect of drugs on

driving ability].

In: Wagner, K., et al., eds. *Handbuch der Verkehrsmedizin: unter Berücksichtigung aller Verkehrswissenschaften*. [Handbook of traffic medicine: with consideration of all traffic sciences]. Berlin: Springer, pp. 854-883 (122 ref.), 1968.

G – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – DC (sensit.) – med.-leg. – mot. vehic. – humans – mammals – blood lev. – other drug lev. – mot. perform. – absorp., distrib., stor. – cardiovasc. – CNS – metab. proc. – analg., antipyret. – autocoids – hormones, hormone antag. – sed., hypnot. – stimulants – *CAAAL-0 B-0457.

The effects of a wide variety of drugs on driving ability are discussed in considerable detail. Simultaneous ingestion of drugs and alcohol is frequent. It has never been shown that drugs can significantly influence the blood alcohol level or the blood alcohol curve, but synergistic effects are well documented for several drug groups, e.g., barbiturates. Although the influence of drugs on alcohol elimination has not been shown, it has been proved that alcohol can considerably affect the elimination of drugs from the body. This may have considerable importance, with respect to prolonged adverse effects of drugs on driving ability.

1284. Soehring, K., and Schüppel, R.

INTERACCION ENTRE ALCOHOL Y MEDICAMENTOS. [Interaction between alcohol and drugs].

Archivos de Biología y Medicina Experimentales (Santiago de Chile), 6: 80-85 (34 ref.), 1969.
Sp – general – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – mammals – blood lev. – species or sex diff. – absorp., distrib., stor. – CNS – metab. proc. – skel., muscle, skin – amphetamines – sed., hypnot. – *CAAAL-0 B-0458.

The subject of alcohol-drug interactions, and experimental evidence to date are reviewed. The importance of the need for study in this area is stressed. The various methods of study—evaluation of clinical data, psychological tests in humans, psychopharmacological studies in animals, and biochemical and pharmacodynamic studies in vitro and in vivo are discussed in detail. It is noted that surprisingly little clinical data from reliable observations have been compiled. Most observations are to be found in unpublished opinions, conclusions, and reports of the pharmaceutical industry; these indicate that interactions with alcohol are particularly likely with the following compounds: hypnotics or anti-epileptics, psychotropic drugs (including antidepressants), isoniazid and other monoamine oxidase inhibitors, antihistamines, oral antidiabetics, analgesics of the morphine type, and pyrazolones.

1285. Soehring, K.

ALKOHOL UND ARZNEIMITTEL. [Alcohol and drugs].

Arch. Pharm. (Weinheim), 303(1): 25 (2 ref.), 1970.

G – abst. – general – DC (add., infra-add., unspec. incr.) – humans – mammals – psychol. perform. – metab. proc. – tranquilizers – *CAAAL-0 B-0561.

The author discusses the importance of alcohol-drug interaction in traffic medicine. Since 20 billion marks are annually spent on alcohol in Germany, and 5 billion marks are spent on drugs, the possibilities of interaction are obvious. The psychological method has been used to investigate meprobamate-alcohol and librium-alcohol reactions, for example, but the real value of such experiments is small, because large differences exist between the observed sample group and the population at large. Despite research, there is no drug which will reliably decrease the blood alcohol concentration. Experiments are discussed which show that simultaneous alcohol administration inhibits drug metabolism, e.g., amidopyrine, 5-hydroxytryptamine, and acetanilid. By blocking the drug oxidation, a conjugation of glucuronic acid or sulfate is stimulated. These findings are unexpected, since alcohol is metabolized through the cytosol ADH system, whereas drugs are metabolized by the microsomes.

1286. Sofronov, N. S.

EKSPERIMENTAL'NOE IZUCHENIE FARMAKOLOGICHESKIKH SREDSTV SONNOI TERAPII: SOOBShCHENIE 1. SNOTVORNOE DEISTVIE BARBAMILA, GEDONALA, NARKOLANA, KHLORALGIDRATA, ETILOVOGO ALKOGOLIA I DVOINYKH SOCHETANII IZ NIKH. [Experimental investigations of pharmacological agents employed in sleep therapy. Report 1. The effect of barbamil, hedonal, narcolan, chloral hydrate, ethanol, and their binary combinations on sleep].

Akademiia Nauk SSSR, Institut Fiziologii imeni I. P. Pavlova, Trudy (Moscow), 1: 243-250 (3 ref.), 1952.

R – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – anesthetics – barbiturates – *CAAAL-0 A-1131.

In tests with barbamil (20 mg/kg) and ethanol (0.5 ml/kg, 96%), neither substance administered alone in the given dose induced sleep in rabbits. Ethanol and barbamil combined manifested a clearly-defined additive synergism—the animals slept 11-19 min. Narcolan (30 mg/kg) plus 0.5 ml/kg ethanol was not soporific in the 5 rabbits tested. The combined effect of these substances in higher doses (100 mg/kg and 1.5 ml/kg, respectively) induced 12-31 min sleep, whereas narcolan alone induced a mean sleeping time of 10 min. It is concluded that a definite synergism was shown between narcolan and ethanol.

1287. Sögnen, E.

APPARENT DEPRESSION IN THE ABSORPTION OF STRYCHNINE, ALCOHOL AND SULPHANILAMIDE AFTER ORAL ADMINISTRATION OF SODIUM FLUORIDE, SODIUM OXALATE, TETRACEMIN AND SODIUM PHYTATE.

Acta Pharmacol. (Copenhagen), 22: 8-18 (17 ref.),

1965.

E – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – G.I. tract – anti-infectants – coagulants – elect., water-bal. agents – stimulants – unclass. ther. agents – *CAAAL-11149-A2 B-0459.

The effect of administration of calcium-binding substances on the blood alcohol concentration (BAC), 1 hr after oral administration of ethanol (1.6 g/kg in 16% sol), was studied in 20 male albino rats. The mean control BAC of 0.89°/oo (8 rats) was lowered to a mean of 0.31°/oo (8 rats) by 50 mg of sodium fluoride/kg, to 0.22°/oo (2 rats) by 400 mg of tetracemin/kg, and to 0.30°/oo (2 rats) by 150 mg of sodium oxalate/kg—i.e., to 35, 25, and 34% of the control BAC, respectively. The results are believed to be due to a nonspecific absorption-delaying action of the calcium-binding substances.

1288. Solms, H.

DIE BEHANDLUNG DER AKUTEN ALKOHOLVERGIFTUNG UND DER AKUTEN UND CHRONISCHEN FORMEN DES ALKOHOLISMUS. [Treatment of acute alcohol poisoning and the acute and chronic forms of alcoholism].

In: Gruhle, H. W., et al., eds. *Psychiatrie der Gegenwart: Forschung und Praxis. II*. Berlin: Springer, pp. 295-339 (239 ref.), 1960.

G – general – DC (antidotal) – humans – drug-dep. humans – psychol. perform. – CNS – metab. proc. – anti-infectants – elect., water-bal. agents – gastrointest. agents – sed., hypnot. – unclass. ther. agents – *CAAAL-0 A-1132.

Reference is made to treatments for acute alcohol intoxication and acute chronic forms of alcoholism in the literature. For accelerating the decomposition of alcohol, drop-infusions of fructose, 10% in a dose of 100 cc, are recommended. Among suggested treatments are high doses of vitamin B₆, insulin (10-20 units 3 times/day im followed 1 1/2-2 hr later by 1 liter milk), glucose (100 g), amino acid, plasma infusions in more serious cases, and antibiotics for delirium tremens. A schematic diagram is given, representing a classification of the most important characteristic points of therapy for chronic alcoholism. Essentially, the therapeutic points may be summed up as follows: detoxication with

hormones and vitamins; use of sedatives, muscle relaxants, and neuroleptics; restoration of liquid and nutritional balance; removal of the craving for alcohol; aversion treatment with apomorphine or emetine; disulfiram treatment for producing an artificial intolerance to alcohol; and psychotherapy.

1289. Solms, W.

BEMERKUNGEN ZU DER ARBEIT VON N. WÖLKART: „IST DIE VERABREICHUNG VON PARALDEHYD BEI ALKOHOLIKERN KONTRAINDIZIERT?“ [Remarks on the work of N. Wölkart: “Is the administration of paraldehyde contraindicated in alcoholics?”]. Wiener Archiv für Psychologie, Psychiatrie und Neurologie (Vienna), 4: 148-150 (0 ref.), 1954. G – general – DC (add., infra-add., unspec. incr.) – humans – CNS – liver, kidney – metab. proc. – sed., hypnot. – *CAAAL-7250-N16 A-1133.

The author acknowledges that Wölkart (Wiener Archiv für Psychologie, Psychiatrie, und Neurologie (Vienna), 4: 145-148, 1954) pointed out the possible dangers of paraldehyde, but there is no better or safer medication for calming the excited patient in acute intoxication. Barbiturates are less safe. Until something better is found, paraldehyde must be used. It is indispensable in severe alcoholic agitation; patients have been seen to die in such conditions if they are not calmed. It is true that liver damage causes slower oxidation of this, as well as other drugs. The comparison Wölkart makes with the action of disulfiram is rather dubious; the mechanism of action is quite different. Wölkart's 2 cases do not show with certainty that death was due to paraldehyde.

1290. Somers, G. F.

PHARMACOLOGICAL PROPERTIES OF THALIDOMIDE (α -PHTHALIMIDO GLUTARIMIDE), A NEW SEDATIVE HYPNOTIC DRUG. Brit. J. Pharmacol. (London), 15: 111-116 (19 ref.), 1960. E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – sed. hypnot. – *CAAAL-0 A-1445.

The effects of thalidomide were investigated in mice, rats, rabbits, cats, and guinea pigs. The following actions were evaluated: acute toxicity, subacute toxicity, motor activity, narcotic activity, movement coordination and holding reflexes, anticonvulsant activity, analgesic activity, hyperthermic and antipyretic activities, autonomic nervous system, heart, respiration, gastro-intestinal tract, urinary system, and interactions with barbiturates, alcohol, reserpine, chlorpromazine, methylamphetamine, and methylphenidate. The possible potentiation of the toxic effects of ethanol was studied by determining the LD₅₀'s of alcohol in groups of mice given different doses of thalidomide. Thalidomide (0, 200, 400, 600, and 800 mg/kg ip) increased the toxicity of ethanol (6-10 g/kg po). The increase in alcohol toxicity was linearly related to the dose of thalidomide.

1291. Spranger, M.

BEEINTRÄCHTIGUNG DER VERKEHRSSICHERHEIT DURCH BARBITURAT-MEDIKATION UND DURCH DIE KOMBINATION BARBITURAT/ALKOHOL. [Impairment of traffic safety by barbiturate medication and by the combination barbiturate/alcohol]. Dissertation, Medical Faculty of the University of Munich, West Germany, 32 pp. (21 ref.), 1964. G – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – barbiturates – *CAAAL-0 A-1134.

The potentiating effect of thiobutabarbitol on alcohol was studied in 52 human subjects, in order to determine their driving ability 24 hr after the narcosis. The subjects received 200 mg/kg thiobutabarbitol po. 6 hr after their blood drug level was tested, 1/2 l of beer was given to each subject; 30 min later, the subjects were given the writing, number, Romberg, finger-nose, and walking-on-white-line

tests. In a supplementary experiment, some subjects received 1 g thiobutabarbital iv, and, after beer ingestion, underwent the same tests. The results revealed a definite potentiating effect after the iv thiobutabarbital narcosis. Therefore, out-patients should be warned against alcohol intake for 24 hr after oral, and at least for 48 hr after iv, thiobutabarbital narcosis.

1292. Spreng, R. W. E.

TOLSEROL IN ACUTE ALCOHOLISM.

J. Nerv. Ment. Dis. (Baltimore), 118: 545-551 (0 ref.),

1953.

E – exp. cont. – DC (antidotal) – humans – acute admin. – in vivo – mot. perform. – psychol. perform. – CNS – musculoskel. agents – *CAAAL-0 A-1135.

Tolserol, 1 g/2 hr, was administered orally, in addition to the regular program of therapy, at the Keeley Institute in Dwight, Ill. The degree of intoxication produced by tolserol was rated by observation. Initially, there was a slightly higher degree of intoxication in acute alcoholics (86 patients, as compared with a control group of 48 patients), but the degree of intoxication soon declined sharply, and intoxication was relieved, on the average, 1 day earlier than in the control group. The symptom of excitement responded more slowly than that of intoxication, but the response was as definite and favourable as was the response to intoxication.

1293. Sroka, K. H.

VON GEWERBLICHEN GESUNDHEITSSCHÄDEN DURCH ALKOHOL. [Occupational health hazards due to alcohol].

Gesundheit und Wohlfahrt (Zurich), 31: 186-194 (0 ref.),

1951.

G – FS – general – DC (add., infra-add., unspec. incr.) – humans – absorp., distrib., stor. – metab. proc. – cardiovasc. agents – miscellaneous – unclass. ther. agents – *CAAAL-5854-H3 A-1136.

The potential danger of ethanol is discussed in the light of its ability to facilitate the absorption of many poisonous substances to which workers are exposed in industry. These hazardous materials include: mercury, lead, arsenic, cyanamide, tetrachlorates, and nitroglycerin. The effect of cyanamide, for example, is 30 times greater in the presence of alcohol. Ethyl and methyl alcohol are also discussed as toxic agents in their own right. Antidotes to poisoning in industry are discussed, as well as the prevention of its occurrence through education.

1294. Stacchini, C.

ÉTUDE CRITIQUE ET EXPÉRIMENTALE SUR L'ANTAGONISME ENTRE LA STRYCHNINE ET L'ALCOOL. [Critical and experimental study on the antagonism between strychnine and alcohol].

Archives de Physiologie Normale et Pathologique (Paris), (Ser. 2) 4: 479-524⁵ (9 ref.),

1877.

F – exp. cont. – DC (decrease) – mammals – other org. – acute admin. – in vivo – dose resp. – cardiovasc. – CNS – stimulants – *CAAAL-0 A-1137.

A number of cases involving the influence of alcohol on strychnine intoxication, of strychnine on alcohol intoxication, and of alcohol on traumatic tetanus (due to the observed similarity between strychnine poisoning and traumatic tetanus) are reviewed. In 21 experiments on frogs, guinea pigs, and dogs, the author investigated: fatal strychnine dosages, dosages of alcohol necessary to produce anesthesia or death, effects of strychnine on animals previously anesthetized by alcohol, and effects of alcohol following strychnine injections. He concludes that alcohol does not act on strychnine like a true antagonist, but does effect a notable diminution of the convulsive manifestations of strychnine poisoning, and can prevent death; it is probably the least dangerous remedy. Strychnine does not appear to influence alcoholic intoxication, nor does it affect fatal alcohol doses. Completely toxic doses of both substances together are fatal in their combined effects.

1295. Staehelin, J. E.

DIE BEDEUTUNG DER SOGENANTEN WECK-AMINE FÜR DIE NEUROLOGIE UND PSYCHIATRIE. [The importance of the so-called Weck-amines for neurology and psychiatry].

Schweiz. Med. Wschr. (Basel), 71(42): 1197-1202 (25 ref.),

1941.

G – SEC – general – DC (decrease) – humans – blood lev. – CNS – amphetamines – *CAAAL-0 A-1138.

The literature on the cerebral amine stimulants is reviewed, and the effects and side effects are discussed. It appears to have been established that pervitin can, under certain conditions, be successful in sobering inebriates for a short time, but the blood alcohol level is not influenced by the drug. Furthermore, it seems probable that the euphoria caused by pervitin leads to a disregard for danger, to higher driving speeds, and, consequently, to an increased accident rate.

1296. Stančák, A.

KRIVKA PRACOVNEJ VÝKONNOSTI V BOURDONOVEJ SKÚŠKE POD VPLYVOM ALKOHOLU A DEXFENMETRAZÍNU. [The effect of alcohol and dexphenmetrazine on the performance efficiency curve in the Bourdon test].

Activ. Nerv. Sup. (Prague), 5(2): 189-190 (5 ref.),

1963.

C – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – psychol. perform. – CNS – stimulants – *CAAAL-11532-J1 A-1139.

80 soldiers, 19-20 yr old, were divided into 4 groups of 20—group A was a control; group B received 20 mg dexphenmetrazine po, and was tested after 25 min; group C received 210 ml 42% alcohol (in form of vodka) plus, 65 min later, 20 mg dexphenmetrazine po, and was tested 90 min after the alcohol; and group D was tested 90 min after 210 ml alcohol. Alcohol significantly lowered performance. Dexphenmetrazine relieved fatigue caused by alcohol, but also produced pathological impairment of mental concentration.

1297. State of California, County of Santa Clara, Department of the District Attorney, Laboratory of Criminalistics

DRUGS IN DRINKING DRIVER CASES.

Laboratory of Criminalistics, Department of the District Attorney, Santa Clara County, San Jose, California, 7 pp. (0 ref.),

1967.

E – stat. surv. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – blood lev. – CNS – analg., antipyret. – anti-infectants – autocoids – elect., water-bal. agents – hormones, hormone antag. – nutritive agents – sed., hypnot. – *CAAAL-0 B-0461.

In 1966, the extent of drug use by drinking drivers in arrests in Santa Clara County, California, was studied. Of 3,409 cases of drinking driver arrests, 703, or 21%, when questioned indicated some kind of drug use; of these 703 cases, 683 different drugs were named. 31% of the drugs named included ataractics and ataxics (19.3%), sedatives and hypnotics (7.5%), and analgesic narcotics (3.8%). 180 drinking driver cases with blood alcohol levels of up to and including 0.15% were arbitrarily screened for barbiturates, meprobamate, glutethimide, and chlordiazepoxide; 1 or more of these drugs were found in 35 or 19.4% of the 180 cases.

1298. State of California, Department of the Highway Patrol

A REPORT ON ALCOHOL, DRUGS AND ORGANIC FACTORS IN FATAL SINGLE VEHICLE TRAFFIC ACCIDENTS (FINAL REPORT).

Department of the Highway Patrol, State of California, U.S.A., 120 pp. (24 ref.),

1967.

E – SEC – stat. surv. – DC (unspec.) – mot. vehic. – humans – blood lev. – barbiturates – hormones, hormone antag. – stimulants – tranquilizers – *CAAAL-0 B-0460.

An extensive and detailed study was made of the influence of alcohol, drugs, and organic factors on single-vehicle accidents in the State of California from November 1963 to October 1965. Of the 102 persons showing positive drug test reactions, 32 or 31.4% had a blood alcohol level of 0, and 6 or 60.8% had a blood alcohol level of .10 or greater. It was impossible to determine the effect of the alcohol-drug combinations from the study data available; however, it was considered certain that drivers using drugs had little hesitation about taking alcohol.

1299. Staub, H.

BEITRÄGE ZUM ANTABUSE-PROBLEM: I. DER EINFLUSS VON ÄTHYLALKOHOL AUF DEN SAUERSTOFFVERBRAUCH WEISSER MÄUSE VOR UND NACH BEHANDLUNG MIT THIURAMDISULFID-DERIVATEN. [Contributions to the antabuse problem: I. The influence of ethyl alcohol on the oxygen consumption of white mice before and after treatment with thiuramdisulfide derivatives].

Helv. Physiol. Pharmacol. Acta (Basel), 13: 121-140 (80 ref.),

1955.

G – ES – exp. comp. – DC (sensit.) – mammals – acute admin. – chronic admin. – in vivo – dose resp. – respir. – unclass. ther. agents – *CAAAL-7381-B2 A-1319.

To study the combined effect of ethanol and thiuramdisulphide derivatives, white mice received: 1) bis (ethylphenyl) thiuramdisulfide (5.0 mg/g), bis (methylphenyl) thiuramdisulfide (5.0-6.0 mg/g), dimorpholylthiuramdisulfide (1.0-1.5 mg/g), dipiperidylthiuramdisulfide (0.8-1.0 mg/g), tetraethylthiuramdisulfide (1.0-2.0 or 3.0-4.0 mg/g), or tetramethylthiuramdisulfide (0.5-0.6 mg/g); 2) an emulsifier plus the thiuramdisulfide compounds, or 3) ethanol (4.5-5.0 mg/g po) plus the thiuramdisulfide compounds. The oxygen consumption was measured. Statistically, the tetramethyl-, tetraethyl-, and dimorpholyl-derivatives significantly increased oxygen consumption, in descending order of potency. The ethylphenyl derivative lowered, and the methylphenyl derivative increased, consumption, but the effect was not significant. The emulsifier, "tween-20", significantly increased oxygen consumption. Ethanol significantly increased oxygen consumption at first, and then later caused inhibition (except for the dimorpholyl derivative, which already, by itself, had caused strong inhibition). The last results are in contrast with the anticipated increase of oxygen consumption caused by the production of acetaldehyde. It is suggested that the results are due to a lesion by the thiuramdisulfide compounds on either a part of the enzyme system of alcohol metabolism, or on all of the liver dehydrogenases.

1300. Staub, H.

BEITRÄGE ZUM ANTABUSE-PROBLEM: 2. DER EINFLUSS VON ÄTHYLALKOHOL AUF DEN SAUERSTOFFVERBRAUCH WEISSER MÄUSE VOR UND NACH BEHANDLUNG MIT DITHIOCARBAMINSÄUREN. [Contributions to the antabuse problem: 2. The influence of ethyl alcohol on the oxygen consumption of white mice before and after treatment with dithiocarbamic acids].

Helv. Physiol. Pharmacol. Acta (Basel), 13: 141-155 (44 ref.),

1955.

G – ES – exp. comp. – DC (sensit.) – mammals – acute admin. – in vivo – dose resp. – respir. – unclass. ther. agents – *CAAAL-7381-B2 A-1320.

The influence of dithiocarbamic acids on the oxygen consumption of white mice was investigated in a 2-phase experiment. In the first phase, the mice received 1% sc solutions of 0.2-0.3 mg/g dimethyldithiocarbamic acid, 0.04 mg/g piperidyl-dithiocarbamic acid, 0.2-0.3 mg/g morpholyldithiocarbamic acid, or a 2% sc sol of 0.5 mg/g diethyldithiocarbamic acid. The dose response curves and oxygen consumption were determined. In the second phase, the mice were given 4.5-5 mg/g alcohol directly into the stomach. The changes in the oxygen consumption of the mice in each phase were again tabulated and graphically evaluated. The effect of the dithiocarbamic acids on the oxygen consumption of white mice was qualitatively the same as that of the corresponding thiuramdisulfide derivatives, the values for which were obtained in a previous study (Helv. Physiol. Pharmacol. Acta, 13: 121-140, 1955). The former, however, were absorbed faster. Since the dithiocarbamic acids are known

to increase the blood acetaldehyde level, it is concluded that their formation in vivo is the cause of the symptoms of the antabuse-alcohol reaction. Due to their ability to form complexes with heavy metals, the acids can inhibit the enzyme systems concerned with alcohol metabolism by chelating heavy metals necessary for enzyme functioning.

1301. Steinhoff, C.

WIRKUNGSÄNDERUNGEN DER KOMBINATION PSYCHOPHARMAKA-PENTOBARBITAL DURCH SUBCHRONISCHE ALKOHOLDARREICHUNG. [Changes in the effect of the combination of psychopharmacological drugs and pentobarbital by the subchronic administration of alcohol].

Dissertation, Medical Faculty of the University of Hamburg, West Germany, 55 pp. (127 ref.), 1966.

G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – CNS – analg., antipyret. – anti-infectants – barbiturates – stimulants – tranquilizers – *CAAAL-0 B-0462.

In guinea pigs, the application of 20 mg/kg ip pentobarbital led to a narcosis of 105 min. After sub-chronical ingestion of 10% ethanol in the drinking liquid, the narcosis was shortened to 58 min. 14 days after stopping the ethanol ingestion, the narcosis time returned to normal. Premedication with 10 mg/kg chlorpromazine im extended the sleeping time by 28% to 136 min. Sub-chronical alcohol ingestion shortened the sleeping time. 4 days after stopping ethanol, the sleeping time was reduced to 129 min. Premedication with 0.2 mg/kg reserpine im, 48 hr before the pentobarbital administration, followed by 0.1 mg/kg im 24 hr later, increased the sleeping time by 31% to 137 min. Sub-chronical alcohol application reduced the reserpine effect from 12 to 17%. The author discusses the different theories which try to explain this effect of alcohol on the action of the 3 drugs.

1302. Steinhoff, D., and Marquardt, P.

KOMBINATION VON KALIUMPYROSULFIT UND ÄTHYLALKOHOL IM TRÄNKUNGSVERSUCH AN RATTEN. [Combination of potassium pyrosulfite and ethyl alcohol in drinking experiments on rats].

Arzneimittelforschung (Aulendorf), 13(3): 237-238 (12 ref.), 1963.

G – ES – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. – in vivo – CNS – metab. proc. – *CAAAL-10570-D2 A-1140.

80 rats were divided into 3 drug groups and 1 control group, each group consisting of 10 males and 10 females. In different experiments, the rats received 30-75 ml/kg 7.5% ethanol and 15-60 mg/kg potassium pyrosulphite, either separately or together. The combined administration of ethanol and potassium pyrosulphite did not produce a degree of toxicosis in excess of that produced by the substances individually.

1303. Stepanov, A. V., and Vifliantsev, N. M.

OPYT ISPOL'ZOVANIIA ETILOVOGO SPIRTA DLIA LECHENIIA OTRAVLENII METANOLOM. [Experience in the use of ethyl alcohol for the treatment of methanol poisoning].

Voенно-Meditsinski Zhurnal (Moscow), 2: 75 (1 ref.), 1970.

R – general – DC (antidotal) – humans – acid-base, blood pH, elect. – metab. proc. – alcohols – *CAAAL-0 B-0992.

The use of alcohol in the treatment of 3 cases of methanol poisoning is reported. 3 young men, who had each drunk about 500 ml of a liquid which was 65% methanol, were admitted to hospital with typical methanol poisoning symptoms: headache, dizziness, sensitivity to light, pain in the eyes, weakness, coated tongue, etc. Methanol was identified in the urine. After their stomachs had been

pumped, the patients received 500 ml of a 5% ethanol sol iv twice a day for 3 days, followed by administration of 50 ml of a 20% ethanol sol every 4 hr for 4 days. Also administered were 250 ml of a 5% bicarbonate of soda sol iv, a 40% glucose sol iv, a 0.5% novacaine sol iv, and a 5% vitamin B₁ sol sc. All symptoms of poisoning gradually disappeared, and the patients were dismissed 12-18 days after the start of treatment. It is concluded that, for treatment to be as effective as in the cases described, large quantities of ethanol must be administered at an early stage of the poisoning.

1304. Stephan, L.

UNTERSUCHUNGEN ÜBER DIE EIGNUNG VON "ACTIVIT" UND "CHOKO AUS MILCH" ALS ERNÜCHTERUNGSMITTEL. [Investigation of the effectiveness of "activit" and "choko-milk" as sobering agents].

Dissertation, Medical Faculty of the University of Heidelberg, West Germany, 117 pp. (61 ref.), 1961.

G – exp. cont. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – mot. perform. – psychol. perform. – absorp., distrib., stor. – metab. proc. – *CAAAL-0 A-1141.

A series of controlled experiments with human subjects was carried out to determine the effectiveness of the commercial preparations, "activit" and "choko-milk", as sobering agents, following ingestion of various quantities of cognac, beer, and wine, under Bourdon, ring, and reaction time test conditions. The tabulated data indicate that activit inhibited the absorption of alcohol without accelerating alcohol metabolism. The alcohol-impaired performance was insignificantly improved in 2 out of 6 tests. Choko-milk inhibited and reduced the absorption time of alcohol, but did not affect alcohol metabolism or psychomotor performance. It is concluded that neither of the agents tested is a suitable antagonist to alcohol.

1305. Stern, L., Kassil, G., and Lokchina, E.

EFFET DE L'ALCOOL ET DU CO SUR LE PASSAGE DU BISMUTH DU SANG DANS LE LIQUIDE CÉPHALO-RACHIDIEN. [Effect of alcohol and CO on the passage of blood bismuth in the cephalorrhachidian liquid].

C.R. Soc. Biol. (Paris), 97: 648-650 (0 ref.), 1927.

F – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – other drug lev. – absorp., distrib., stor. – CNS – miscellaneous – *CAAAL-0 A-1142.

Rabbits were acutely intoxicated by 6-10 cc/kg 10% alcohol, or, in 1 case, by 30 cc/kg ethanol. Chronic poisoning was induced by the addition of 10 g/kg alcohol in the feed for 13-18 days. Bismuth subnitrate, 0.16-0.24 g/kg ip or iv, was then administered. After acute, chronic, or subacute poisoning, the presence or absence of bismuth in the cephalorrhachidian fluid or in the nerve tissue was determined. In most cases, bismuth was detected simultaneously in the fluid and the nerve tissue; in a smaller number of cases, bismuth was found in the fluid, but not in the tissue, or vice versa. It was concluded that alcohol weakens the resistance of the hematoencephalic barrier to bismuth. Similar tests were made, and results similar to those with alcohol were produced, with carbon monoxide.

1306. Stern, M. M.

ANTI-HISTAMINE TREATMENT OF ALCOHOLISM.

J. Nerv. Ment. Dis. (Baltimore), 122: 198-199 (5 ref.), 1955.

E – SEC – exp. – case hist. – cross-tol. – humans – psychot. humans – drug-dep. humans – chronic admin. – in vivo – CNS – metab. proc. – autocoids – *CAAAL-7545-M3 A-1321.

The use of an antihistamine in the treatment of 11 alcoholics is reported. Each patient received 50 mg of pyrilamine maleate 3 times/day po for 1-21 days. All 11 patients reported a loss of desire for compulsive drinking. 1 patient stopped drinking immediately after receiving 0.5 cc of pyrilamine maleate iv. Another patient reported an unaccustomed craving for sweets and better tolerance of

alcohol. All 11 patients eventually relapsed to compulsive drinking. The author suggests that compulsive drinking, once initiated, may continue, because of secondary autonomic and metabolic imbalance, such as a backfeeding mechanism for the production of histamine-like metabolites. An histamine antagonist, such as pyrilamine maleate, may break up the cycle by minimizing the effects of the metabolites. Other experimenters have reported a decreased craving and an increased tolerance for alcohol in histamine-treated schizophrenics, and a beneficial use of ACTH and adrenocortical hormones in treating alcoholics. It is concluded that antihistamines can be used in the temporary treatment of compulsive drinking.

1307. Stessel', T. A.

ISSLEDOVANIYA O KOMBINIROVANNOM DEISTVII NARKOTIKOV. SOOBSHCHENIE I. KOMBINIROVANNOE DEISTVIE PAROV NARKOTIKOV NA BELYKH MYSHEI.

[Investigations on the combined effects of narcotics: I. Combined effect of narcotic vapours on white mice].

Fiziol. Zh. S.S.S.R. Sechenov (Moscow), 22: 129-138 (10 ref.),

1937.

R – GS – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – alcohols – anesthetics – miscellaneous – unclass. ther. agents – *CAAAL-0

A-1143.

White mice were subjected to poisoning by vapours of narcotic mixtures: acetone plus methanol, methanol plus cyclohexane, methanol plus toluene, acetone plus toluene, acetone plus benzene, benzene plus methanol, acetone plus cyclohexane, toluene plus cyclohexane, ethyl ether plus acetone, ethyl ether plus methanol, or ethanol plus methanol, employing Fühner's method. The minimum concentration of each substance, alone and in combination, necessary to evoke a light narcosis (leaning to the side) was determined. The results are presented graphically, according to Loewe's method. The author concludes that an additive effect of the individual narcotics in combination may be assumed. To substantiate this assumption, in vitro experiments were carried out in a subsequent report by the same investigator (Fiziol. Zh. S.S.S.R. Sechenov., 22: 247-251, 1937).

1308. Stessel', T. A.

ISSLEDOVANIE O KOMBINIROVANNOM DEISTVII NARKOTIKOV. SOOBSHCHENIE II. O GEMOLIZE IN VITRO PRI DEISTVII SMESEI NARKOTIKOV. [Investigations on the combined effects of narcotics: II. Hemolysis in vitro under the effect of various indifferent narcotic compounds].

Fiziol. Zh. S.S.S.R. Sechenov (Moscow), 22: 247-251 (3 ref.),

1937.

R – GS – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – in vitro – blood comp., sites, lymph – alcohols – anesthetics – miscellaneous – sed., hypnot. – *CAAAL-0

A-1144.

This is a sequel to an earlier study in which white mice were subjected to vapours of different narcotic mixtures (Fiziol. Zh. S.S.S.R. Sechenov, 22: 129-138, 1937). In this work, in vitro experiments were carried out on the erythrocytes of rabbits, with various binary mixtures (methanol plus acetone, ethanol plus acetone, methanol plus ethanol, ether plus chloroform, ethanol plus chloroform, urethane plus chloral hydrate, and ethanol plus chloral hydrate). The min hemolytic concentration of each substance employed in the test was determined, and then the concentrations for each binary mixture, giving 100%, more than 100%, and less than 100% of the hemolytic concentration, were established. The tests were carried out at room temperature. The results are presented graphically, according to Loewe's method. The results indicate that the effect of the mixtures is almost strictly additive. The results of 5 component mixtures (methanol-ethanol, chloroform, acetone, and ethyl ether) give also a simple addition effect.

1309. Stevens, H., and Forster, F. M.
 EFFECT OF CARBON TETRACHLORIDE ON THE NERVOUS SYSTEM.
 Arch. Neurol. (Chicago), 70: 635-649 (84 ref.), 1953.
 E – general – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – absorp., distrib., stor. – CNS – G.I. tract – liver, kidney – anti-infectants – *CAAAL-0 A-1145.

The effect of carbon tetrachloride (CCl_4) on the nervous system is described in detail. Acute CCl_4 poisoning is dominated by symptoms of the central nervous system, such as headache, diplopia, incoordination, paresthesia, impaired vision, confusion, and coma. An etiological classification is tabulated. 15 cases of CCl_4 poisoning are reported, and typical cases are described. Neuropathological findings are given, and the literature is reviewed. It is pointed out that 11 of the 13 adult patients encountered by the author were alcoholics, and, moreover, had been drinking alcoholic beverages immediately before, during, or soon after CCl_4 exposure. Considering the problem of alcohol enhancement of CCl_4 toxicity, it is noted that alcohol may increase the rate and degree of absorption in the gastrointestinal tract, but reinforcement of the toxic effects occurs in animals and humans even when the CCl_4 is inhaled. Preexisting alcoholic cirrhosis in humans may play some role, but this is not a factor in animal experiments. The available evidence appears to indicate that the coexistence and combined effect of alcohol ingestion and CCl_4 poisoning is not due merely to chance.

1310. Stewart, R. D., Torkelson, T. R., Hake, C. L., and Erley, D. S.
 INFRARED ANALYSIS OF CARBON TETRACHLORIDE AND ETHANOL IN BLOOD.
 J. Lab. Clin. Med. (St. Louis), 56(1): 148-156 (7 ref.), 1960.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – anti-infectants – *CAAAL-9534-B2 A-1146.

4 groups of 5 or 6 rabbits were exposed to carbon tetrachloride (CCl_4) vapours (2,500 or 5,000 parts/million). Immediately prior to exposure, 2 of these groups received 3 ml alcohol/kg po, and a fifth group received only alcohol. Blood samples of CCl_4 , alcohol, and serum glutamic oxalacetic transaminase (SGOT) were taken at intervals 1.5-8.5 hr after exposure. The CCl_4 concentrations were significantly higher in rabbits exposed to alcohol plus the 5,000 parts/million CCl_4 dosage; concentrations were also higher in animals given the lower CCl_4 dose, but the difference was not significant. SGOT levels were higher in animals exposed to both compounds; after 4 hr, the SGOT levels were twice as high in rabbits given alcohol plus CCl_4 as in those given CCl_4 alone, but, after 8.5 hr, the difference was of questionable significance. Whereas alcohol directly or indirectly increased the average concentration of CCl_4 in the blood, CCl_4 failed to affect the average blood alcohol concentration.

1311. Steyn, D. G.
 THE USE OF PARALDEHYDE IN ALCOHOLIC DELIRIUM TREMENS.
 Med. J. Aust. (Sydney), 40(2): 91-93 (30 ref.), 1953.
 E – SEC – general – cross-tol. – DC (add., infra-add., unspec. incr.) – CNS – liver, kidney – sed., hypnot. – *CAAAL-6848-N6 A-1147.

The use of paraldehyde in the treatment of alcoholic delirium tremens is reviewed and contraindicated for the following reasons: alcoholics are tolerant to the drug's sedative effects, and at times paraldehyde may increase restlessness; paraldehyde may aggravate delirium tremens, instead of tranquilizing the patient; and, because of the advanced liver condition in alcoholics, they possess an increased susceptibility to the toxic action of paraldehyde. As well, owing to the synergistic pharmacological and toxicological effects of paraldehyde and alcohol, the use of paraldehyde is discouraged.

1312. Stiefbold, E. -G.
*EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE KONZENTRATION
 BLUTZUCKERSENKENDER SULFONAMIDE IN DER LEBER UND DEREN EINFLUSS*

AUF DIE IM ALKOHOLSTOFFWECHSEL WICHTIGEN ZWISCHENSUBSTANZEN.

[Experimental investigations of the concentration of sulfonamides in the liver which reduce the blood sugar level, and their influence on important intermediate products of alcohol metabolism].
Dissertation, Medical Faculty of the University of Heidelberg, West Germany, 20 pp. (13 ref.),

1959.

G – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp.
– liver, kidney – *CAAAL-0 A-1148.

In a series of controlled experiments, the alcohol metabolites adenosine triphosphate, lactic acid, and pyruvic acid were determined in the livers of healthy rats, before and after treatment with nadisan. At therapeutic doses of nadisan (400 mg/kg) over a prolonged period of time, there was no accumulation in the liver. Conversely, toxic doses (1000 mg/kg) resulted in accumulation. No nadisan was detected in the mitochondria. Therapeutic doses over an 8-14 day period reduced the adenosine triphosphate and pyruvic acid, and increased lactic acid production. Toxic doses over an 8-day period decreased adenosine triphosphate, lactic acid, and pyruvic acid.

1313. Stolman, A.

COMBINED ACTION OF DRUGS WITH TOXICOLOGICAL IMPLICATIONS—PART 1.

Progress in Chemical Toxicology (New York), 3: 305-361 (134 ref.), 1967.

E – review – DC (add., infra-add., unspec. incr.) – humans – mammals – blood lev. – CNS – metab. proc. – analg., antipyret. – anticonvulsants – barbiturates – miscellaneous – sed., hypnot. – stimulants – tranquilizers – *CAAAL-0 B-0993.

Aspects considered are: factors affecting the duration of drug action, the decreased duration of combined drug action, and the increased duration of combined drug action (the last category embracing volatile anesthetics, ethanol, sedatives and hypnotics, and miscellaneous sedatives, analgesics, anticonvulsants, and muscle relaxants). The concentration and absorption of ethanol, and its terminal concentrations are discussed, and an extensive review is made of its interaction with: paraldehyde, barbiturates, tranquilizers, analgesics, stimulants, depressants, and miscellaneous drugs (quinine, β -dimethylamino-ethyl benzilate, serotonin, SKF-183A, asparagine, and carbon monoxide).

1314. Störmer, A., and Kautzsch, E.

BEHANDLUNG BEI ALTERS- UND GEFÄSSERKRANKUNGEN:

REGULATIONSABLAUFE BEI DER BEHANDLUNG MIT CUMARIN. [Treatment of geriatric and vascular diseases: action of coumarin during treatment].

Therapiewoche (Karlsruhe), 4: 590-594 (0 ref.),

1954.

G – SEC – general – DC (add., infra-add., unspec. incr.) – humans – drug-dep. humans – blood comp., sites, lymph – *CAAAL-0 A-1149.

A general discussion is given on treatment with coumarin, and its reaction mechanism. One case history is reported in which alcohol use during anticoagulant therapy affected clotting factors in the blood, and caused bleeding. A 24 yr-old alcoholic under coumarin treatment experienced a petechial efflorescence on the extremities, and increased bleeding activity of the mucosa. The cause of the condition was found to be a deficiency in factor VII. The patient observed that, after ingestion of beer or wine, the petechiae were readily manifested. An alcohol test under controlled conditions substantiated the observations made by the patient—the factor VII level in the oxalate plasma had decreased by 45%.

1315. Streichenberger, G., Boismare, F., Guy, J., and Lechat, P.

ESSAI DE DIFFÉRENCIATION ENTRE SYNERGIE ADDITIVE ET

POTENTIALISATRICE DANS LE CAS D'ASSOCIATIONS D'HYPNOTIQUES. [Attempt at differentiation between the additive and potentiating synergism of combinations of hypnotics].

Thérapie (Paris), 23: 175-189 (8 ref.),

1968.

F – ES – SEC – exp. comp. – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mammals
 – acute admin. – in vivo – dose resp. – mot. perform. – CNS – *CAAAL-0 B-0562.

The existing definitions of additive and potentiative synergism are reviewed, and a definition is proposed which more precisely establishes the direction of enhancement of effects, since synergism may not occur reciprocally between the 2 members of a drug combination (i.e., drug X may enhance the effect of drug Y, but the reverse does not occur). The method was employed, using the following combinations: pentobarbital-secobarbital, pentobarbital-clomethiazole, penthiobarbital-clomethiazole, clomethiazole-chloral, and chloral-alcohol. Groups of 10 rats weighing 17-23 g received ethanol (3.80, 3.85, 3.95, 4, or 4.5 g/kg) or chloral (250, 275, 300, 350, or 400 mg/kg), and sleeping-time dose-response curves (DRC) were plotted. Then, 45, 60, 80, and 100% of the minimum effective dosage (MED) of alcohol was combined with 45% of the MED of chloral (124 mg/kg), and the DRC was plotted; a similar DRC was plotted for 45% of the MED of ethanol (1.71 g/kg) plus chloral. It was found that the chloral-ethanol combination produces an additive synergism--partial addition when increasing doses of ethanol are added to 45% of the MED of chloral, and complete addition when increasing doses of chloral are added to 45% of the MED of ethanol; the DRC in the former case approximates the theoretical additive DRC, while the DRC in the latter case can be superimposed on the theoretical additive DRC. This indicates that ethanol exercises a predominant action, since increasing doses of ethanol added to a fixed dose of chloral exhaust the effect of chloral, in direct proportion to the amount of ethanol added.

1316. Strongin, E. I.

TOLERANCE AND ANTAGONISM AS MANIFESTED WITHIN THE HUMAN BODY UNDER THE INFLUENCE OF CAFFEINE, CIGARETTES, AND ALCOHOL.

Ph.D. Thesis, Graduate School of Cornell University, Ithaca, N.Y., U.S.A., 96 pp. (28 ref.),

1935.

E – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – mot. perform. – CNS – G.I. tract – *CAAAL-719-D1 A-1150.

Investigated were the development of tolerance to caffeine and cigarette smoke, the effect of coffee, alcohol, and both combined on parotid secretion and motor control, and the antagonism of coffee and cigarette smoke. In 1 series of tests, human subjects were given 1-1 1/2 pints of coffee or caffeine in water (totalling 2-3 grains of caffeine), 40-100 cc alcohol (sufficient amount to induce a measurable reaction), or both substances in combination. Parotid gland secretion and motor control (steadiness and motor coordination) were measured. Alcohol inhibited gland secretion, and coffee stimulated it. When alcohol and coffee were taken together, there was an antagonistic reaction, and secretion approximated normal for 1 hr after coffee ingestion, after which the coffee effect wore off, and the alcohol inhibition was again apparent. If coffee was taken after alcohol inhibition had set in, the antagonism was less intense and of shorter duration. Alcohol disturbed steadiness and motor coordination, and coffee had a slightly beneficial effect. The combined dose produced an antagonistic reaction, with the coffee effect dominant, and steadiness and motor coordination almost normal; the effect lasted for at least 1 hr with regular coffee drinkers, and 2 or more hr with non-users. Also reported in abridged form in Strongin, E. I., and Winsor, A. L., *Journal of Abnormal and Social Psychology* (Washington), 30: 301-313, 1936.

1317. Strongin, E. I., and Winsor, A. L.

THE ANTAGONISTIC ACTION OF COFFEE AND ALCOHOL.

Journal of Abnormal and Social Psychology (Washington), 30: 301-313 (9 ref.),

1936.

E – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – mot. perform. – CNS – G.I. tract – *CAAAL-719-D1 A-1151.

A series of tests on human subjects studied the effects of alcohol, caffeine, and both combined on parotid gland secretion and motor coordination. Measurement of the effects on secretion showed that

coffee antagonized the alcohol inhibition for about 1 hr. When coffee was taken after the alcohol inhibition had firmly set in, its antagonism was less intense, and of shorter duration. With respect to motor coordination, there was an antagonistic reaction after the coffee-alcohol combination, with the coffee effect dominant, and motor coordination practically normal for at least 1 hr in a coffee-user and for at least 2 hr in a non-user. Also reported in more detail in Strongin, Edward Israel, Ph. D. Thesis, Graduate School of Cornell University, Ithaca, N.Y., U.S.A., 96 pp. (28 ref.), 1935.

1318. Sugihara, N.

UEBER DEN PANAX GINSENG. NR. 1. [On *Panax ginseng*. I].

Keijo Journal of Medicine (Keijo), 1: 347-408 (46 ref.),

1930.

G – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – dose resp. – CNS – *CAAAL-0 A-1152.

A brief history of *Panax ginseng*—its origin, growth, extraction, and general attributes—is given. Its interaction with various substances, including alcohol, was investigated. Controlled experiments with alcohol and *Panax* established that the min sleep-inducing dose of alcohol was 80 mg/10 g in rats given *Panax*, and 50 mg/10 g in controls. *Panax*-treated rats all survived alcohol doses of 20 mg, and there were 2 deaths at 30 mg; the corresponding results in controls were 1 and 3 deaths respectively. The period until the onset of sleep was relatively longer, and sleeping time was shorter with *Panax* pretreatment. The min lethal dose of alcohol was 120 mg/10 g in *Panax*-treated rats and 90 mg in the controls. In addition to lower mortality values with *Panax*, the onset of death was relatively more prolonged than in the controls.

1319. Sullivan, G. A.

DUAL MEDICATION IN ACUTE ALCOHOLISM.

G.P. (Kansas City), 9: 67-69 (8 ref.),

1954.

E – exp. cont. – DC (antidotal) – drug-dep. humans – acute admin. – in vivo – CNS – musculoskel. agents – *CAAAL-7353-N11 A-1153.

111 intoxicated patients were given daily doses of mephesisin (5 g) and sedamyl (1.3 g). An identical control group of 74 patients received mephesisin (20 cc iv initially, and 4.0 g po per day thereafter) and barbital (average dosage 0.6 g on the first day, followed by routine reduction thereafter). It is concluded that the sobering-up period can normally be shortened by oral therapy—80% of the study group were sober within 36 hr, compared with 62% of the controls. Use of barbiturates can be completely eliminated in routine cases, and iv therapy may normally be avoided.

1320. Sulser, F., Watts, J., and Brodie, B. B.

ANTAGONISTIC ACTIONS OF IMIPRAMINE (TOFRANIL) AND RESERPINE ON CENTRAL NERVOUS SYSTEM.

Fed. Proc. (Bethesda), 19: 268 (1 ref.),

1960.

E – abst. – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – CNS – antidepressants – tranquilizers – *CAAAL-0

A-1154.

Experiments were conducted to investigate whether reserpine-induced depression might serve as a model to study the antidepressant action of imipramine. Reserpine (2.5 mg/kg ip) given to rats prolonged the narcosis elicited by alcohol (3 g/kg) from the control time of 5 min to about 120 min. In animals pretreated with imipramine (25 mg/kg ip), 3 to 6 hr before administration of reserpine, the potentiation was almost completely blocked. In contrast, imipramine did not inhibit the alcohol potentiation induced by chlorpromazine.

1321. Sulser, F., Watts, J., and Brodie, B. B.

ON THE MECHANISM OF ANTIDEPRESSANT ACTION OF IMIPRAMINELIKE DRUGS.

Ann. N.Y. Acad. Sci. (New York), 96: 279-288 (19 ref.), 1962.
 E – SEC – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
 – in vivo – dose resp. – CNS – antidepressants – tranquilizers – *CAAAL-10281-D2 A-1155.

Adult male mice received 2.5 mg/kg reserpine ip, followed 1 hr later by 3 g/kg ethanol ip; average sleeping time was 150 ± 12 min. When 25 mg/kg imipramine was added 10 min before reserpine administration, sleeping time averaged 120 ± 14 ; however, the same dose of imipramine blocked the potentiation of subthreshold doses of ethanol when given 3 hr before reserpine (sleeping time— 27 ± 2 min). 10.0 mg/kg desmethyylimipramine also blocked potentiation (sleeping time— 13 ± 3 min).

1322. Sunshine, I., Hodnett, N., Hall, C. R., and Rieders, F.

PREVALENCE OF DRUGS AND/OR CARBON MONOXIDE IN VICTIMS OF FATAL VEHICULAR ACCIDENTS.

Fourth International Meeting in Forensic Medicine, Copenhagen, Denmark, 17 pp. (14 ref.),

1966.

E – presentation – stat. surv. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – blood lev. – other drug lev. – blood comp., sites, lymph – sed., hypnot. – tranquilizers – *CAAAL-0 B-0463.

The results of blood and urine analyses of specimens obtained from victims of fatal vehicular accidents in Philadelphia, Pennsylvania, and Cuyahoga County (Cleveland), Ohio, are reported. Each blood sample was analyzed to determine if ethanol and other volatiles, carbon monoxide, barbiturates, meprobamate, or glutethimide were present. Urine samples were screened for salicylates, phenothiazines, glucose, acetone, and organic bases (including amphetamine). 31 accident victims were found to have a carboxyhemoglobin (COHb) level of 5-10%, and 13 had a level of 11-20%. Of these, 4 persons had a level of 11-20% COHb and a blood alcohol concentration (BAC) of 0.01-0.05 g/100 ml; 5 had a COHb level of 5-10% and a BAC of 0.06-0.10 g/100 ml, 1 had a COHb level of 11-20% and a BAC of 0.06-0.10 g/100 ml, 4 had a COHb level of 5-10% and a BAC of 0.11-0.15 g/100 ml, 8 had a COHb level of 5-10% and a BAC of 0.17 g/100 ml or more, and 6 had a COHb level of 11-20% and a BAC of 0.16 g/100 ml or more. The effects of ethanol on a person who has absorbed some carbon monoxide were not studied, but an additive effect is presumed.

1323. Sunshine, I., Hodnett, N., Hall, C. R., and Rieders, F.

DRUGS AND CARBON MONOXIDE IN FATAL ACCIDENTS.

Postgrad. Med. (Minneapolis), 43(3): 152-155 (16 ref.),

1968.

E – stat. surv. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – blood lev. – other drug lev. – blood comp., sites, lymph – barbiturates – *CAAAL-0 B-0464.

The results of blood and urine analyses of specimens obtained from victims of fatal vehicular accidents in Philadelphia, Pennsylvania, and Cuyahoga County (Cleveland), Ohio, are reported. Each blood sample was analyzed to determine if ethanol and other volatiles, carbon monoxide, barbiturates, meprobamate, or glutethimide were present. Urine samples were screened for salicylates, phenothiazines, glucose, acetone, and organic bases (including amphetamine). 31 accident victims were found to have a carboxyhemoglobin (COHb) level of 5-10%, and 13 had a level of 11-20%. Of these, 4 persons had a level of 11-20% COHb and a blood alcohol concentration (BAC) of 0.01-0.05 g/100 ml; 5 had a COHb level of 5-10% and a BAC of 0.06-0.12 g/100 ml; 1 had a COHb level of 11-20% and a BAC of 0.06-0.10 g/100 ml; 4 had a COHb level of 5-10% and a BAC of 0.11-0.15 g/100 ml; 8 had a COHb level of 5-10% and a BAC of 0.16 g/100 mg or more, and 6 had a COHb level of 11-20% and a BAC of 0.16 g/100 ml or more. The effects of ethanol on a person who has absorbed some carbon monoxide were not studied, but an additive effect is presumed.

1324. Süss, W.

ZUR GENIESSBARKEIT DES FALTENTINTLINGS (*COPRINUS ATRAMENTARIUS*).[The edibility of the inky cap (*Coprinus atramentarius*)].

Zeitschrift für Pilzkunde (Heilbronn), 15: 54-56 (2 ref.),

1936.

G – exp. comp. – DC (sensit.) – humans – acute admin. – in vivo – cardiovasc. – respir. – *CAAAL-0 A-1446.

The author reviews reported poisoning symptoms which developed after the combined ingestion of the mushroom, *Coprinus atramentarius*, and alcohol, and a self-experiment is described. In all known cases in which *Coprinus* was combined with alcohol, poisoning symptoms such as the following occurred: accelerated pulse (130-150), red flushing or rubor of the face, neck, and shoulders, a strong thirst, dry mouth, difficulty in breathing and speaking, staggering, and a pounding of the heart. To confirm these effects, the author on 2 occasions consumed about 10 teaspoonsful of the mushroom (presumably cooked) without alcohol, and found no ill effects. On a third occasion, at 8:00 pm, he took the same amount of mushrooms, together with 300 ml beer. At 10:40, he experienced a feeling of heat, a flushed face, and a pulse of 100. By 11:30, the symptoms began to abate, and by midnight of the following day, upon his drinking a glass of cider and a cup of coffee with brandy, the symptoms returned in a milder form. It is concluded that *Coprinus* has toxic effects when taken with or before alcohol. It is therefore only conditionally edible, and should not be sold to the public.

1325. Sutherland, V. C., Hine, C. H., and Burbridge, T. N.

THE EFFECT OF ETHANOL ON CEREBRAL CORTEX METABOLISM *IN VITRO*.

J. Pharmacol. Exp. Ther. (Baltimore), 116: 469-479 (28 ref.),

1956.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – mammals – in vitro – species or sex diff. – CNS – metab. proc. – antispasmodics – elect., water-bal. agents – *CAAAL-7680-B1 A-1156.

The effects of 0.056 M ethanol on the metabolism of rat and human cerebral tissue respiring in the presence of glucose, pyruvate, acetate, succinate, glutamate, or potassium chloride plus glucose were studied, using conventional manometric and chemical methods. The oxygen uptake of the rat cerebral cortex was increased by ethanol in the presence of all substrates except succinate; however, oxygen uptake of the human cerebral cortex was not increased in the presence of any substrate. Ethanol had no effect on glucose uptake by rat or human cortex, nor did it affect lactic acid accumulation in rat cortex in the presence of glucose, pyruvate, or acetate. In the presence of succinate and glutamate, lactic acid accumulation was reduced by ethanol. With human cerebral cortex, ethanol did not affect lactic acid accumulation in the presence of glucose, but reduced lactic acid accumulation in the presence of pyruvate. Utilization of extracellular lactic acid was affected by ethanol only in the presence of glutamate. Ethanol slightly decreased oxygen uptake in potassium chloride-stimulated cortex; glucose uptake and lactic acid accumulation were both increased by ethanol.

1326. Sutherland, V. C., Burbridge, T. N., Adams, J. E., and Simon, A.

CEREBRAL METABOLISM IN PROBLEM DRINKERS UNDER THE INFLUENCE OF ALCOHOL AND CHLORPROMAZINE HYDROCHLORIDE.

J. Appl. Physiol. (Bethesda), 15: 189-196 + 10 unpublished tables (31 ref.),

1960.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – drug-dep. humans – acute admin. – in vivo – blood lev. – blood comp., sites, lymph – cardiovasc. – CNS – metab. proc. – tranquilizers – *CAAAL-0 A-1157.

Cerebral blood flow and metabolism were studied in male human subjects who had been problem drinkers for an average of 11 yr. In the first test, 1 g/kg alcohol was administered po as bourbon whiskey diluted with an equal vol of water. In the second test, the above alcohol dose was given after 7 days of pretreatment with chlorpromazine hydrochloride (25 mg im on the first day, followed by successively increased po doses to a total of 400 mg/day for the last 2 days). Blood samples and

electroencephalograms were taken. It was found that the chronic use of alcohol produced an alteration in the metabolism of glutamic acid in the CNS and in the content of glutamic and lactic acids in the arterial blood. Chlorpromazine administration for a week affected more cerebral and peripheral changes than have been established for a single dose. Despite a multiplicity of effects with alcohol, including a pronounced change in behaviour after chlorpromazine, the values for cerebral oxygen and glucose consumption were normal.

1327. Svedin, C. -O.

TISSUE DISTRIBUTION OF CHLORMETHIAZOLE AND COMPATIBILITY WITH ETHANOL AND CERTAIN DRUGS.

Acta Psychiat. Scand. (Copenhagen), 42(Suppl. 192): 27-34 (3 ref.), 1966.
E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – sed., hypnot. – *CAAAL-0 B-0465.

The tissue distribution, excretion, metabolism, teratogenic effect, and compatibility with ethanol, promazine, pentobarbital, and promethazine were investigated in rats, mice, and rabbits. In 1 test, ethanol was injected ip into mice 10 min before the administration of different doses of chlormethiazole, and the dose at which a hypnotic effect (as shown by a loss of righting reflex for at least 30 sec) could be obtained in 50% of the animals (HD₅₀) was estimated by determination of loss of righting reflex. An additive effect, but no potentiation, was noted on the HD₅₀.

1328. Szücs, J., and Kisch, B.

ÜBER DIE KOMBINIERTE WIRKUNG VON FLUORESZIERENDEN STOFFEN UND ALKOHOL. [The combined effects of fluorescent compounds and alcohol].

Z. Biol. (Berlin), 58: 558-570 (17 ref.), 1912.
G – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – other org. – acute admin. – in vivo – anti-infectants – *CAAAL-0 A-1158.

Controlled experiments were carried out on the combined effect of a fluorescent substance (e.g., eocene, 2 1/2 cc) and alcohol (sol of 1 1/2-10%) at 24°C on *Colpidium colpoda* cultures. The light sources employed were a small Nernst lamp and a Osram lamp of 50 candle power. It was found that the combination of even a low alcohol sol (2 1/2%) and a photodynamic substance (0.2 cc eocene) showed appreciable synergism in light as well as in the dark. Alcohol (0.1 cc, 10%) and methylene blue (0.5 cc) showed no synergistic effect in the dark. Alcohol, added to low concentrations of methylene blue which had been inactive in light for some hr, induced a ready extinction of the cultures, although the alcohol amounts added did not affect the cultures when introduced alone. Similar results were obtained in all 12 tests (at constant light under controlled and consistent test conditions).

1329. Tacker, M., Creaven, P. J., and McIsaac, W. M.

ALTERATION IN TYRAMINE METABOLISM BY ETHANOL.

Biochem. Pharmacol. (New York), 19(2): 604-607 (6 ref.), 1970.
E – exp. cont. – mammals – acute admin. – in vivo – dose resp. – other drug lev. – absorp., distrib., stor. – metab. proc. – autonomic agents – *CAAAL-0 B-0994.

Fasted male Sprague-Dawley rats (200 g) received 2 g/kg ethanol as a 25% sol ip, followed 20 min later by tyramine-1¹⁴C HBr (1.9 mg/kg, 5.3 mc/m-mole in 0.5 ml saline) ip. A control group received ethanol only. Urine was collected at intervals up to 96 hr, and the metabolites in the urine were then determined. It was found that ethanol pretreatment causes no significant difference, either in the rate of excretion of radioactivity, or in the total amount excreted in urine. Ethanol does cause a 16% decrease in the amount of free and conjugated p-hydroxyphenylacetic acid-1¹⁴C excreted, the effect being most marked (42%) in the excretion of p-hydroxyphenylacetyl glycine-1¹⁴C. There is a 20-fold

increase in tyrosol- ^{14}C excretion, the most noticeable effect being found in tyrosol sulfate. Free tyrosol and free and conjugated N-acetyltyramine are unaltered by ethanol. The decrease in free and conjugated p-hydroxyphenylacetic acid and the increase in conjugated tyrosol represent 14.99% and 15.12% of injected radioactivity, respectively. The results indicate that tyramine metabolism is altered by ethanol in the same way as is that of serotonin and norepinephrine. A specific inhibition of ethanol on glycine conjugation is suggested.

1330. Takemori, A. E.

STUDIES ON MORPHINE-ADAPTED CEREBRAL CORTICAL SLICES OF RATS.

Fed. Proc. (Bethesda), 21(2): 177 (0 ref.),

1962.

E – abst. – exp. cont. – cross-tol. – DC (unchanged) – mammals – chronic admin. – in vitro – CNS – barbiturates – hallucinogens – *CAAAL-10282-B2 A-1159.

In experiments on rats, it was found that pentobarbital (2×10^{-4} M), ethanol (0.4 M), and morphine (1×10^{-3} M) depressed the respiratory rate of cortical slices from control animals to about the same degree. In morphine-adapted cortical slices, pentobarbital and ethanol produced a marked depression of the stimulated respiratory rate, thus showing that the slices are not cross-adapted to these agents. Nalorphine failed to prevent the depression by pentobarbital and ethanol, at concentrations which prevented the effect of morphine in vitro.

1331. Tamburrini, N.

DEGLI ANTAGONISTI DELLA STRICNINA E SPECIALMENTE

DELL'ALCOOL—CONTRIBUZIONE CRITICO-SPERIMENTALE. [On the antagonists of strychnine, especially alcohol—critico-experimental contribution].

Giornale Internazionale delle Scienze Mediche (Naples), 1: 757-769 (0 ref.),

1879.

I – exp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – blood comp., sites, lymph – cardiovasc. – CNS – stimulants – *CAAAL-0 A-1160.

The effect of alcohol as an antidote to strychnine was studied in a series of comparative experiments in which rabbits were given, respectively, strychnine sulphate in doses of 5 mg in 1 g distilled water together with 1 g 90% alcohol, 1 g 90% alcohol followed by 8 mg strychnine sulphate in 1 g distilled water 4 min later, and 8 mg strychnine sulphate administered prior to alcohol. The following conclusions were derived: alcohol is not an antagonist to strychnine and proved incapable of inducing tolerance to triple the strychnine dose. Alcohol showed similar effects, whether administered simultaneously with or after strychnine. Nevertheless, alcohol decreased and ameliorated tetanic phenomena, and prolonged life.

1332. Tammisto, T.

INCREASED TOXICITY OF 5-HYDROXYTRYPTAMINE BY ETHANOL IN RATS AND MICE.

Scand. J. Clin. Lab. Invest. (Oslo), 21(Suppl. 101): 89 (0 ref.),

1968.

E – abst. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – CNS – metab. proc. – *CAAAL-0 B-0466.

Young adult rats and mice were given 5-hydroxytryptamine (5-HT) through the tail vein, 30 min after ip administration of ethanol. In the rats, ethanol caused a 40-fold increase in the toxicity of 5-HT during anesthesia, with a mean survival time of 15 min. In mice, the increase in toxicity during ethanol anesthesia was only 3-fold, and the mean survival time was more than 3 hr. It is concluded that ethanol causes only a minor increase in the effect of iv 5-HT in mice. In rats, the increase in toxicity by ethanol involves mechanisms seen during general anesthesia, and others possibly due to inhibition of 5-HT metabolism.

1333. Tang, P. C., and Rosenstein, R.

THE INFLUENCE OF ALCOHOL AND DRAMAMINE, ALONE AND IN COMBINATION, ON PSYCHOMOTOR PERFORMANCE.

U.S. Naval Aerospace Medical Institute and U.S. Army Aeromedical Research Unit, Pensacola, Florida, Report No. NAMI-1002, 16 pp. (6 ref.) March 7, 1967.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – gastrointest. agents – *CAAAL-0 B-0467.

The effects of alcohol and dramamine, alone and in combination, were studied in 8 experiments on the performance of 4 young adult subjects on the Scow complex coordinator. Alcohol alone produced a 12.5% decrease in performance when the blood alcohol level was between 44 and 50 mg%. When the blood alcohol decreased to the 35 mg% level, the performance impairment became insignificant. Dramamine alone (100 mg) produced a relatively small performance impairment (max 6%). The combination of alcohol and dramamine produced relatively large performance impairments—during the first 3 hr following ingestion of dramamine and alcohol, the performance decrements were 8%, 25%, and 9%, respectively, when the blood alcohol levels were 50, 44, and 34 mg%, respectively. The reasons for not recommending a maximum permissible alcohol level for airmen are discussed.

1334. Tang, P. C., and Rosenstein, R.

INFLUENCE OF ALCOHOL AND DRAMAMINE, ALONE AND IN COMBINATION, ON PSYCHOMOTOR PERFORMANCE.

Aerospace Med. (St. Paul), 38(8): 818-821 (6 ref.),

1967.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – gastrointest. agents – *CAAAL-0 B-0468.

The effects of alcohol and dramamine, alone and in combination, were studied in 8 experiments on the performance of 4 young adult subjects on the Scow complex coordinator. Alcohol alone produced a 12.5% decrease in performance when the blood alcohol level was between 44 and 50 mg%. When the blood alcohol decreased to the 35 mg% level, the performance impairment became insignificant. Dramamine alone (100 mg) produced a relatively small performance impairment (max 6%). The combination of alcohol and dramamine produced relatively large performance impairments—during the first 3 hr following ingestion of dramamine and alcohol, the performance decrements were 8%, 25%, and 9%, respectively, when the blood alcohol levels were 50, 44, and 34 mg%, respectively. The reasons for not recommending a max permissible alcohol level for airmen are discussed.

1335. Tara, M. S.

OXYCARBONÉMIE ET ÉTHYLÉMIE. [Carbon monoxide and blood ethanol levels].

Arch. Mal. Prof. (Paris), 14: 60-63 (0 ref.),

1963.

F – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – blood comp., sites, lymph – indust. intox. – *CAAAL-0 A-1161.

The results of observations on 23 automobile plant workers exposed to carbon monoxide (CO) fumes under the influence of alcohol and tobacco are presented. 37% (7 workers) showed positive blood alcohol values at high CO concentrations. Blood alcohol levels rose appreciably at lunch time, and dropped sharply 4 hr after lunch. The ratios of alcohol to CO in the blood are tabulated. A higher CO concentration in the blood among drinkers is indicated. No relationship was found between CO in the blood and time away from work. Conversely, the duration of work suspension had a marked influence on alcohol absorption. Alcohol, rather than other factors such as work duration, is the aggravating factor in CO poisoning.

1336. Tarsitano, F.

INTERFERENZE TRA ALCOOL E FENILISOPROPILAMINA DAL PUNTO DI VISTA MEDICO-LEGALE. [Interference between alcohol and phenylisopropylamine from a

medico-legal point of view].

Boll. Soc. Ital. Biol. Sper. (Naples), 20: 370-371 (0 ref.),

1945.

I – abst. – exp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – CNS – metab. proc. – amphetamines – stimulants – *CAAAL-0 A-1460.

The results of studies on dogs, conducted by V.M. Palmieri of the University of Naples, concerning the effects of amphetamine and desoxyephedrine on the determination of blood alcohol and on the blood alcohol curve, are reported. It has been found that po administration of amphetamine does not cause the appearance of volatile reducing agents in the blood which could interfere with blood alcohol tests. Amphetamine and desoxyephedrine, whether administered simultaneously with alcohol or at the peak of the alcohol effect, do not cause any variations in the blood alcohol curve. It is suggested that the mitigation or prevention of the clinical symptoms of inebriation produced by the amines should be attributed, not to their influence on alcohol metabolism, but to their action on the same nerve sites as are affected by alcohol.

1337. Taylor, J. D., Wilson, L., Nash, C. W., and Cameron, D. F.

THE EFFECTS OF ETHYL ALCOHOL AND AMPHETAMINE ON PERFORMANCE.

Canadian Federation of Biological Societies, Proceedings (Montreal), 7: 36 (0 ref.),

1964.

E – abst. – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – amphetamines – nutritive agents – *CAAAL-0

A-1162.

32 students were subjected to a series of tests to determine the combined effect of alcohol and amphetamine on performance. The alcohol (sufficient rye whiskey to provide 1.2 g ethyl alcohol/kg) was combined with either 15 mg amphetamine plus 300 mg lactose or with a 300 mg lactose placebo. Intercorrelations under 4 test conditions (lactose, amphetamine, alcohol-lactose, and alcohol-amphetamine) ranged from 0.189 to 0.531. When amphetamine was administered together with alcohol, mental addition was improved, compared to the same task performed at equivalent blood alcohol levels without amphetamine. It is concluded that amphetamine appears to decrease test-retest reliability, and that alcohol tends to counteract this effect of amphetamine.

1338. Teare, R. D.

SOME PROBLEMS OF BARBITURATE AND ALCOHOLIC INTOXICATION.

Medicoleg. J. (Cambridge), 34, Part 1: 4-10 (19 ref.),

1966.

E – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – absorp., distrib., stor. – liver, kidney – barbiturates – *CAAAL-11955-D1 B-0469.

Barbiturate, alcohol, and barbiturate-alcohol intoxications are discussed. The explanation for ingestion of the enormous doses of barbiturates and alcohol found in the mortality statistics, particularly if there is no suicidal intent, is difficult to find. On the average, 15 g of a rapidly-acting barbiturate and almost 1/3 of a bottle of whiskey appear to be ingested. "There is no doubt that many alcoholics tend to counteract the withdrawal effects of alcohol with barbiturates, while not infrequently the two drugs are combined in an attempt to achieve an effect which surpasses those of either. Whether confusion initiated by alcohol can lead to the reckless ingestion of barbiturates, or vice versa, it is impossible to say, but it may be that some if not all of these cases fit into the ... definition of accidental or unintentional suicide." The author concludes that, "A great deal has yet to be learned by chemical analysis of blood and urine for alcohol, barbiturates, tranquilizers, etc., as to the role of these agents in accidents—be they road traffic or domestic. Alcohol is not a factor in the deaths of drivers or passengers of two-wheeled vehicles. While the effects of alcohol and barbiturate are probably additive rather than synergistic, the confusion associated with the ingestion of these drugs may lead to unexpected and unintentional self-destruction."

1339. Teger, A. I., Katkin, E. S., and Pruitt, D. G.
EFFECTS OF ALCOHOLIC BEVERAGES AND THEIR CONGENER CONTENT ON
LEVEL AND STYLE OF RISK TAKING.
J. Personality Soc. Psychol. (Washington), 11(2): 170-176 (17 ref.), 1969.
E – exp. comp. – congen. stud. – humans – acute admin. – in vivo – mot. perform. – psychol. perform.
– *CAAAL-0 B-0563.

The authors performed experiments to test the validity of the generally-accepted Cohen hypothesis, that alcohol distorts the perception of the amount of risk taken, but does not affect the willingness to take risk. 36 healthy students, 21 years of age or more, were divided into 2 groups, each group filling out a choice-dilemma questionnaire before and after alcohol consumption. Each subject received 4 doses of alcohol, making a total of 0.8 ml ethanol/kg of body weight, in the form of 3 different alcoholic beverages—synthetic alcohol, vodka, or bourbon, with no, low, and high congener content, respectively. The responses on the questionnaires were evaluated, and the results were tabulated. Only the high-congener beverage showed a significant effect; the effects of the vodka and the pure alcohol tended in the predicted direction, but the results were not significant. The authors conclude that persons drunk on high-congener beverages are more willing to take risks than sober ones or those drunk on low-congener beverages, and that alcohol does affect the willingness to take risk. Risk patterns from other experiments show that young persons or groups will take more risk than older people or individuals.

1340. Teisinger, J.
CHRONISCHE TRINITROTOLUOL-EINWIRKUNG UND DER EINFLUSS VON
ALKOHOL AUF DIE UMWANDLUNG DIESES STOFFES IM KÖRPER. [Chronic effects
of trinitrotoluol and the influence of alcohol on the metabolism of this compound in the body].
Archiv für Gewerbepathologie und Gewerbehygiene (Berlin), 4: 491-499 (7 ref.), 1933.
G – exp. cont. – DC (decrease) – humans – acute admin. – chronic admin. – in vivo – other drug
lev. – cardiovasc. – CNS – metab. proc. – *CAAAL-0 A-1163.

The effect of alcohol was studied in workers in whom notable blood changes had been established prior to the tests, and who were then exposed to trinitrotoluene (TNT) vapours or dust. The 3 subjects were given 1/2 l beer each per experiment, in a series of 3 tests. The tabulated data revealed vasomotor changes and TNT reduction products (positive Websterian reaction) in the urine which could not be detected prior to alcohol intake, and which disappeared from the urine 60 min later. The author conjectures that alcohol is closely related to the metabolism of TNT, and explains the mechanism as follows: TNT is deposited in the tissue of workers, and slowly reacts; alcohol, which accelerates oxidation in tissue, reduces TNT to various intermediates at a highly accelerated rate, with a proportionate effect on the CNS.

1341. Teisinger, J.
CHRONICKÝ VLIV TRINITROTOLUENU A VLIV ALKOHOLU NA PŘEMĚNU TÉTO
LÁTKY V TĚLE. [Chronic effect on the organism of trinitrotoluene poisoning, and its
modification by alcohol].
Cas. Lek. Cesk. (Prague), 72: 645-648 (8 ref.), 1933.
C – exp. cont. – DC (decrease) – humans – acute admin. – chronic admin. – in vivo – other drug
lev. – cardiovasc. – CNS – metab. proc. – *CAAAL-0 A-1164.

The effect of alcohol was studied in workers in whom notable blood changes had been established prior to the tests, and who were then exposed to trinitrotoluene (TNT) vapours or dust. The 3 subjects were given 1/2 l beer each per experiment, in a series of 3 tests. The tabulated data reveal vasomotor changes and TNT reduction products (positive Websterian reaction) in the urine which could not be detected prior to alcohol intake, and which disappeared from the urine 60 min later. The author conjectures that alcohol is closely related to the metabolism of TNT, and explains the mechanism as

follows: TNT is deposited in the tissue of workers, and slowly reacts; alcohol, which accelerates oxidation in tissue, reduces TNT to various intermediates at a highly accelerated rate, with a proportionate effect on the CNS.

1342. Theobald, W., and Stenger, E. G.

ZUR GEGENSEITIGEN WIRKUNGSSTEIGERUNG VON ALKOHOL UND

PSYCHOPHARMAKA. [Reciprocal potentiation between alcohol and psychotropic drugs].

Arzneimittelforschung (Aulendorf), 12: 531-533 (14 ref.),

1962.

G – ES – FS – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – cardiovasc. – CNS – anesthetics – antidepressants – *CAAAL-10318-D2

A-1165.

The effects of alcohol and imipramine in combination were determined in white mice, rats, and cats. In mice, it was found that the toxicity of alcohol in doses between 0.25 and 3 cc/kg was markedly increased by 30 mg/kg imipramine. The mortality from 650 mg/kg imipramine was increased from 47 to 80% by 3 cc/kg alcohol. In rats, narcosis was markedly prolonged when 25 mg/kg imipramine was combined with 3 cc/kg alcohol. In 5 anesthetized cats, 3 mg/kg imipramine produced a marked potentiation of the depressant effect of 1.4 cc alcohol iv.

1343. Theobald, W., Büch, O., Kunz, H. A., Morpurgo, C., Stenger, E. G., and Wilhelmi, G.

VERGLEICHENDE PHARMAKOLOGISCHE UNTERSUCHUNGEN MIT TOFRANIL, PERTOFRAN, UND INSIDON. [Comparative pharmacological investigations with tofranil, pertofran and insidon].

Arch. Int. Pharmacodyn. (Gand), 148(3-4): 560-596 (71 ref.),

1964.

G – ES – FS – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – species or sex diff. – cardiovasc. – CNS – antidepressants – *CAAAL-11156-D2

A-1166.

Central and peripheral effects of tofranil (imipramine), pertofran (desimipramine), and insidon (opipramol), alone or in combination with alcohol, were studied in rats, hamsters, cats, and mice. Narcotic effects were only slightly potentiated when 2-5 ml ethanol/kg were administered po or ip, 30-180 min after one of the drugs. Circulatory carotid modifications in cats showed that, in the mean, the lethal dose of a 20% ethanol infusion was unaffected by pretreatment with one of the drugs. 2 ml ethanol/kg, combined with 30-35 mg/kg of 1 of the drugs in a single injection iv, showed a significantly higher toxicity in mice than that of either substance alone.

1344. Theorell, H., and Yonetani, T.

LIVER ALCOHOL DEHYDROGENASE-DPN-PYRAZOLE COMPLEX: A MODEL OF A TERNARY INTERMEDIATE IN THE ENZYME REACTION.

Biochemische Zeitschrift (Berlin), 338: 537-553 (28 ref.),

1963.

E – exp. cont. – DC (Add., infra-add., unspec. incr.) – mammals – acute admin. – in vitro – liver, kidney – metab. proc. – *CAAAL-0

A-1461.

In vitro tests showed that pyrazole, found to be a potent inhibitor of horse liver alcohol dehydrogenase (LADH), formed a new ternary complex (EPO) with LADH and DPN, characterized by a new absorption maximum at 290 m μ . 1 molecule of LADH bound 2 molecules of pyrazole, a substrate analogue inhibitor. Thus, the actual enzyme reaction proceeds between 1 molecule of LADH, 2 of DPN, and 2 of ethanol. Described are studies on the effect of pH on DPN-binding sites of LADH; dissociation constants of DPN and EPO in EPO complexes; proton liberation effects accompanying the association of LADH, DPN, and pyrazole, and of the zinc ion with pyrazole; and crystallization of the EPO complex. In the case of alcohol, it is probable that the zinc ion causes a dissociation into alcoholate and hydrogen ions. It is suggested (a scheme of reactions is shown) that, in alcohol oxidation by LADH, there is a migration of a hydride ion between carbon-1 of the substrate and

carbon-4 of the pyridine ring of DPN. Although fatty acids compete with ethanol, as does pyrazole, to form strong ternary complexes with LADH and DPN, they give negative complexes at the 290 m μ band. The experiments illustrate how the binding site of substrates is completed only after the coenzyme has been attached to the protein.

1345. Thimann, J.

SEDATION OF ALCOHOLIC PATIENTS WITH NONSEDATIVE DRUGS.

Amer. J. Psychiat. (Hanover), 109: 701-702 (3 ref.),

1953.

E – general – conj. addict. – DC (antidotal) – humans – CNS – autonomic agents – sed., hypnot. – *CAAAL-0 A-1167.

The author reports the results of treating 155 alcoholic patients with the vagus depressant, bellafoline, and the sympathetic depressant, ergotamine. Patients received, in tablet form, between 0.75 and 2 mg bellafoline and between 3 and 8 mg gynergen (ergotamine) daily for 1 to 3 days. The patients seemed to be less tense, and said they felt relaxed. It is concluded that this nonsedative medication may prove useful in acute and subacute stages of intoxication.

1346. Thiry, U.

PATHOLOGIE DES OUVRIERS DE LA CYANAMIDE CALCIQUE INDUSTRIE À POUSSIÈRES. [Pathology of workers affected by calcium cyanamide dust].

Arch. Mal. Prof. (Paris), 4: 132-142 (6 ref.),

1942.

F – SEC – stat. surv. – DC (sensit.) – humans – blood lev. – mot. perform. – cardiovasc. – nerv. syst. – respir. – skel., muscle, skin – miscellaneous – *CAAAL-0 A-1322.

The effects of calcium cyanamide dust on factory workers were studied and compared to those of linen and cotton dust. The harmful effect of calcium cyanamide in combination with alcohol is pointed out—this is attributed to its reaction with alcohol to form diethyl cyanamide (CN₂(C₂H₅)₂). Other explanations which are rejected: 1) nervous system rendered more susceptible by cyanamide; 2) fixation of cystines is hindered; and 3) reduction of glutathion is suppressed. The effects of alcohol plus calcium cyanamide are likened to those of amyl nitrite alone. The author concludes that, by itself, calcium cyanamide is not too toxic, and that it has the advantage of forcing the workers to stay sober. The dust particles and dry heat in the factory are blamed as the major causes of the ill health of some of the calcium cyanamide workers. Their health is found to be somewhat better than that of workers in the cotton and linen industries.

1347. Thomas, J. R.

SNAKE BITES—TREATED BY BRANDY.

Northwestern Medical and Surgical Journal (Chicago), 7: 305-306 (0 ref.),

1855-56.

E – general – DC (antidotal) – drug-dep. humans – *CAAAL-0

A-1168.

A case of snake bite is reported, in which the adult male victim was bitten on the left heel, both fangs having been well inserted into the muscle. Over a period of 36 hr, 1 quart of brandy, 1 1/2 gallons of whiskey, and some mild cathartics were administered. Despite the enormous quantity of alcohol consumed, there was no intoxication; in fact, the patient requested more than was given. After 36 hr, the wounds had healed, and the patient was sound and well. The next day, the patient was seen barefooted, wading in the grass, and feeling with his feet. Asked if he had lost anything, the man replied, "No." When asked again what he was doing, the answer was, "I am hunting a snake. There ain't any liquor only what Doc. Thomas has, and he won't let me have any unless I am snake bit, so I am hunting one." The author advocates the use of brandy or proof spirits of any kind, in large and frequent doses (and recommends that his patients become intoxicated), as well as some mild cathartics, in all cases of bites by poisonous snakes. He adds that he has never been compelled to visit his patients more than twice, and often only once. In an appended note, the editor remarks that, "We

publish the above not for any intrinsic value which it possesses, but because it affords an interesting example of the *strength* of a man's appetite for *strong drink* on the one hand, and a good specimen of the imperfect record of facts on the other."

1348. Thompson, T. O.

SOBERING-UP.

Brit. Med. J. (London), 2: 1294 (1 ref.),

1961.

E – general – DC (antidotal) – humans – *CAAAL-9630-N14

A-1169.

A method of sobering-up a drunk person is described, which is presumed to be the basis of the "prairie oyster" remedy for a hangover. For those who were obviously drunk and incapable when the author was a student, 1 tablespoon of vinegar had a remarkable sobering effect. When used on one extremely drunk man who was in a club, the effect was so swift that he drove his own car home in 20 min.

1349. Tipton, D. L., Jr., Sutherland, V. C., Burbridge, T. N., and Simon, A.

EFFECT OF CHLORPROMAZINE ON BLOOD LEVEL OF ALCOHOL IN RABBITS.

Amer. J. Physiol. (Bethesda), 200: 1007-1010 + 10 unpublished tables (10 ref.),

1961.

E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – autonomic agents – barbiturates – tranquilizers – *CAAAL-9729-A2

A-1170.

Groups of 7-9 rabbits received alcohol by stomach tube (1.56 g/kg) or iv (0.78 g/kg), with or without pretreatment for 7 days with chlorpromazine (6 mg/kg/day sc), reserpine (.4 mg/kg/day sc), or phenobarbital (3 mg/kg/day sc). The above po alcohol dose was also administered in combination with single doses of chlorpromazine (3 mg/kg sc), atropine (2 mg/kg sc), and hexamethonium (2 mg/kg sc). It was found that chlorpromazine elevated the blood alcohol level in rabbits, whether alcohol was given po or iv. Neither reserpine, atropine, or hexamethonium affected the blood alcohol level. Since the above autonomic blocking agents failed to influence the blood alcohol level, and since chlorpromazine did not elevate the blood level of other substances absorbed in a manner similar to ethanol, it is concluded that no part of the chlorpromazine effect is due to an increase in alcohol absorption, but that the effect is probably due entirely to an inhibition of alcohol metabolism.

1350. Tirri, R.

INDUCED TOLERANCE TO PROMAZINE IN MICE AS A PHYSIOLOGICAL ADAPTATION.

Annales Academiae Scientiarum Fennicae, Series A, IV: Biologica (Helsinki), No. 103: 54 pp. (124 ref.),

1966.

E – exp. cont. – cross-tol. – mammals – acute admin. – chronic admin. – in vivo – dose resp. – cardiovasc. – tranquilizers – *CAAAL-0

B-0995.

The characteristics, criteria, and possible mechanisms of induced tolerance to promazine were investigated. In 1 experiment, mice were chronically treated for 3 weeks with daily ip injections of promazine, morphine hydrochloride, ethanol (10% w/v), and amphetamine sulphate. At the end of this time, single ip test injections were given of saline, the same drug, or a different drug. 1 group chronically-treated with 30 mg/kg promazine/day, received 3 g/kg alcohol, and a chronically-treated group given 3 mg/kg alcohol/day received 10 mg/kg promazine. The hypothermic effect was studied using colonic temperature, and the hyperthermic effect was determined using survival time at +45° C as criterion. The chronic alcohol administration caused partial tolerance to the hypothermic and hyperthermic effects of alcohol. Chronic promazine administration did not result in tolerance to alcohol. In preliminary experiments, however, shorter chronic administration (3 and 6 days) of promazine established cross-tolerance to alcohol. A weak tolerance to the hypothermic effect of promazine developed in mice chronically-treated with alcohol. It is concluded that the compensatory

mechanisms which later develop against promazine are so specific that chronic treatment cannot protect against the effects of alcohol. The weak cross-tolerance of alcohol-treated animals to promazine in the hypothermic effect may be considered a secondary phenomenon.

1351. Többen, H.

ÜBER CHLOROFORMSUCHT IN VERBINDUNG MIT ANDEREN SUCHTEN.

[Chloroform addiction in conjunction with other addictions].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 12: 285-291 (23 ref.),

1928.

G – general – conj. addict. – DC (decrease) – drug-dep. humans – CNS – anesthetics – *CAAAL-0 A-1356.

The author studied the influence of chloroform addiction on the use of other similar drugs. The subject of investigation was a pharmacist who had become an alcoholic, and used cocaine to antagonize the alcoholic intoxication. Thereafter, he used cocaine at various times and in varying doses, and he also tried novocaine and eucaine B. Since the latter 2 did not have the expected effect, he transferred to chloroform. Some time later, he again started using cocaine, and consumed considerable amounts of alcohol. He was hospitalized, but, even then, he kept consuming alcohol and using chloroform. The author concludes that the origin of chloroform addiction can be due to previous abusive use of alcohol and/or cocaine or other similar substances. It is also pointed out that other drug addicts, especially morphinists, are susceptible to chloroform addiction, and it is suggested that the licenses of doctors or pharmacists be immediately suspended if they abuse their easy access to drugs.

1352. Toll, N.

LIBRIUM AS AN ADJUNCT TO PSYCHOTHERAPY IN PRIVATE PSYCHIATRIC PRACTICE.

Dis. Nerv. Syst. (Galveston), 21: 264-266 (5 ref.),

1960.

E – SEC – general – DC (add., infra-add., unspec. incr.) – psychot. humans – CNS – tranquilizers – *CAAAL-0 A-1171.

22 men and 43 women received from 30 to 55 mg of librium/day. The therapeutic effect was most marked when the predominant symptoms were tension, anxiety, withdrawal, insomnia, and general irritability. Symptoms of depression, anorexia, and depersonalization were moderately affected, and alcoholism, overweight, impotence, and frigidity were unaffected or only minimally changed. The drug was not only well tolerated, but the patients took it willingly, preferring it to all other tranquilizing drugs they had taken previously. Librium seems to potentiate the effect of alcohol, and therefore it is important to warn the patients to abstain from drinking while taking librium.

1353. Tomaszewska, Z., and Dąbski, H.

ŚMIERTELNE ZATRUCIE GLIMIDEM. [Lethal poisoning with glimide].

Pol. Tyg. Lek. (Warsaw), 21: 1461-1462 (5 ref.),

1966.

Po – ES – RS – general – DC (add., infra-add., unspec. incr.) – post-mort. – humans – blood lev. – other drug lev. – CNS – G.I. tract – liver, kidney – sed., hypnot. – *CAAAL-0 B-0470.

A brief description of glutethimide, which is employed as an effective sedative and hypnotic, is given, and a case history is cited. The subject was found unconscious, following ingestion of 5 g of glutethimide (glimide). The lethal dose of this drug is estimated at 10-20 g. A post-mortem examination (the patient died 20 hr following ingestion of the alcohol and drug) disclosed a blood alcohol level of 0.35°/oo. Chemical analysis disclosed glimide in the blood, urine, stomach, and liver. The authors conclude that the toxicity of glutethimide is appreciably increased by alcohol.

1354. Tomits, G.

NYSTAGMUS VIZSGÁLAT KISMENNYISÉGŰ ALKOHOL, DORLOTYN ÉS ALKOHOL—DORLOTYN ADÁSÁRA. [Nystagmus tests following the administration of small amounts of alcohol, dorlotin, and alcohol with dorlotin].

Fülörgegyezgyaszat (Budapest), 8: 26-30 (7 ref.),

1962.

H – ES – GS – RS – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – nerv. syst. – senses – barbiturates – *CAAAL-10399-D1

A-1172.

Positional and rotation nystagmus were investigated after administration of alcohol, dorlotin (amobarbital), and the 2 drugs in combination. It was found that alcohol and alcohol plus dorlotin are more effective in causing spontaneous nystagmus than dorlotin alone, but the action of dorlotin is more prolonged. Dorlotin and dorlotin plus alcohol are more effective than alcohol in bringing about nystagmus in the looking direction, and the combined action of alcohol and dorlotin is the most durable. In causing positional nystagmus, the most effective drugs are alcohol and alcohol plus dorlotin, the action of the combined dose being the most lasting. Administration of alcohol plus dorlotin most frequently causes bilateral turning of the nystagmus. It is concluded that there is synergism between dorlotin and alcohol; the barbiturate derivatives have been found to intensify the action of alcohol, and combined consumption causes more marked and prolonged effects.

1355. Tonning, D. J.

METHYL ALCOHOL POISONING: A SURVEY OF THIRTY CASES.

Nova Scotia Med. Bull. (Halifax), 24(1): 1-8 (14 ref.),

1945.

E – SEC – DC (unchanged) – humans – blood lev. – acid-base, blood pH, elect. – metab. proc. – alcohols – *CAAAL-4148-C4

A-1173.

This paper reports on 30 cases of acute methanol poisoning, 11 of them fatal. Symptoms and physical and laboratory findings are presented in detail. The vital factor was to combat the acidosis as quickly as possible. Some cases received ethanol by mouth, but this practice was discontinued, because it appeared that the ethyl alcohol was poorly oxidized, the daily blood estimations of ethanol concentration becoming alarmingly high. In general, recovery was uneventful, with no residual pathology demonstrated, except marked eye changes in 1 case. Also reported in Branch, Arnold, and Tanning, D. J., Canad. J. Public Health (Toronto), 36: 147-151, 1945.

1356. Töpken, A.

EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DEN EINFLUSS VON ASPIRIN, VERONAL UND PYRAMIDON AUF BLUTALKOHOLKURVE UND TRUNKENHEIT.

[Experimental investigations on the influence of aspirin, veronal, and pyramidon on the blood alcohol curve and drunkenness].

Dissertation, Medical Faculty of the University of Heidelberg, Germany, 19 pp. (24 ref.),

1938.

G – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – mot. perform. – CNS – analg., antipyret. – barbiturates – *CAAAL-0

A-1174.

In a series of controlled experiments, a study was made of the effects of aspirin (2 x 0.5 g), veronal (0.5 g), and pyramidon (2 x 0.3 g), administered po to human subjects under the influence of alcohol (3/4 liter—8.8%). None of the drugs tested showed any significant influence on the blood alcohol level. On the other hand, all of the 3 substances increased intoxication symptoms, such as fatigue, positive Romberg test, unsteadiness, etc. During the 4 weeks of tests, no essential change in the blood alcohol curve could be observed, in relation to tolerance. Greater skill was achieved with the ring test, due to tolerance. The number of errors increased during periods of fatigue.

1357. Torka, J.

CAVE MEPROBAMATE UND ALKOHOLGENUSS. [Beware of meprobamate when ingesting alcohol].

Munchen. Med. Wschr. (Munich), 103: 896 (0 ref.), 1961.
 G – general – DC (add., infra-add., unspec. incr.) – humans – cardiovasc. – CNS – respir. – analeptics
 – musculoskel. agents – stimulants – tranquilizers – *CAAAL-0 A-1175.

The author recounts the case of a woman who took 70 mg meprobamate over a period of several hr, followed by ingestion of 3-4 glasses of alcoholic punch in the evening. She later collapsed, was found to have ceased to breathe, and was without pulse or reflexes. She was revived by prompt administration of large doses of cardiazole, eukraton, and some cortisone, and recovered within several days. In the author's opinion, the alcohol reacted with the meprobamate to produce an almost abrupt paralysis of the CNS and autonomic nervous centers, and the patient would almost certainly have died without the immediate medical attention which she received.

1358. Torres Orrego, R.
 FISIOPATOLOGIA DE LA INTOXICACION ALCOHOLICA AGUDA EXPERIMENTAL EN EL HOMBRE Y SUS MODIFICACIONES FARMACOLOGICAS. [Physiopathology of acute experimental alcohol intoxication in man and its pharmacological modifications].
 Actas Luso. Esp. Neurol. Psiquiat. (Madrid), 13: 145-151 (17 ref.), 1954.
 Sp – exp. – review – DC (decrease) – DC (add., infra-add., unspec. incr.) – humans – blood lev. – CNS – metab. proc. – barbiturates – cardiovasc. agents – elect., water-bal. agents – hormones, hormone antag. – miscellaneous – nutritive agents – *CAAAL-7018-D3 A-1176.

Research undertaken at the Servicio de Investigaciones Psiquiatricas at Santiago de Chile is reviewed. Studies are discussed in connection with the elimination of alcohol under conditions of hyper- and hypo-glycemia, the action of alcohol in hypoglycemia and insulin coma and the differences in arterial and venous bloods, and the effect of various drugs on alcohol intoxication; reference is made to nicotinamide, somnifene, luminal, and nicotinic acid. 2 types of drugs may be distinguished with respect to effects on acute alcohol intoxication—those which enhance the elimination of alcohol and protect the nervous system from it, and those which aggravate the intoxication.

1359. Tourtellotte, W. W., and Coon, J. M.
 SYNERGISTIC EFFECT OF SODIUM ACETATE AND ETHANOL IN ANTAGONIZING SODIUM FLUOROACETATE POISONING IN MICE.
 Fed. Proc. (Bethesda), 8: 339 (1 ref.), 1949.
 E – abst. – exp. comp. – DC (decrease) – DC (supra-add. incr.) – mammals – acute admin. – in vivo – dose resp. – elect., water-bal. agents – *CAAAL-0 A-1323.

A group of mice was administered sodium fluoroacetate (1080) sc (LD₅₀ of 17.0 g/kg) and then immediately treated ip with ethanol, sodium acetate, or sodium acetate dissolved in ethanol. The most effective antidote for the 1080 poisoning was 2.0-3.0 g/kg (16-24% sol, respectively) of sodium acetate, injected with 1.6 g/kg of ethanol (20% v/v). This combination saved 90-100% of mice poisoned with 10 LD₅₀ of 1080. The immediate treatment of poisoned mice with 1.6 g/kg of ethanol or 3.0 g/kg of sodium acetate increased the LD₅₀ of 1080 by a factor of 3 (54.0 mg/kg) or by a factor of about 4 (63.0 mg/kg), respectively. However, a combination of 3.0 g/kg of sodium acetate dissolved in 1.6 g/kg of ethanol raised the LD₅₀ of 1080 by a factor of 12 (201.0 mg/kg). It is concluded that ethanol and sodium acetate act synergistically in antagonizing 1080 poisoning.

1360. Tracey, J. P., and Sherlock, P.
 HEPATOMA FOLLOWING CARBON TETRACHLORIDE POISONING.
 New York J. Med. (New York), 68(16): 2202-2204 (13 ref.), 1968.
 E – SEC – general – DC (add., infra-add., unspec. incr.) – humans – liver, kidney – anti-infectants – *CAAAL-0 B-0471.

The case history of a patient who died of hepatocellular carcinoma, 7 yr after acute intoxication with carbon tetrachloride (CCl_4), is reported; it is considered to be the first report of hepatoma occurring after CCl_4 exposure. The unusual clinical and laboratory findings suggested necrosis of a large tumor mass. The liver and kidney failure that developed might have been less severe if the patient had not ingested alcohol prior to his exposure to CCl_4 . There is a frequent association of hepatoma with cirrhosis of various types in man, and, in this case, it is impossible to exclude an underlying alcoholic cirrhosis, which is suggested by the long history of moderate alcohol ingestion.

1361. Traquair, H. M.

TOBACCO AMBLYOPIA.

Edinburgh Medical Journal (Edinburgh), 42(2): 153-172 (0 ref.),

1935.

E – SEC – general – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – CNS – nerv. syst. – senses – *CAAAL-2837-H6 A-1177.

According to the author, the data collected concerning the total annual cases of tobacco amblyopia and the total arrests for drunkenness show nothing to indicate that drunkenness is common at the time when tobacco amblyopia occurs, or that alcohol is at all concerned in the production of tobacco amblyopia. "It is quite possible that in other countries alcohol may be a factor, but in these cases, the disease is not due to ethyl-alcohol or to methyl-alcohol, but to toxic distillates, such as those contained in wood spirit, which have found their way through manufacture or sale into alcoholic drinks."

1362. Trenholm, H. L., Wiberg, G. S., and Coldwell, B. B.

ENZYMATIC STUDIES ON THE MECHANISM OF ETHANOL-BARBITURATE INTERACTIONS.

Fifth International Meeting of Forensic Sciences, Toronto, Ontario, Canada, 15 pp. (12 ref.),

1969.

E – exp. cont. – presentation – DC (add., infra-add., unspec. incr.) – mammals – in vitro – liver, kidney – metab. proc. – barbiturates – *CAAAL-0 B-0472.

The biological mechanisms involved in the interaction between alcohol and barbiturates were investigated in rat liver homogenates and hepatic tissue slices. The simultaneous injection of ethanol into live animals produced a dose-dependent decrease in the ip 24-hr LD_{50} for 5 representative barbiturates. The increased toxicity was more pronounced with phenobarbital and barbital—the barbiturates least subject to biotransformation. In vitro studies with tissue slices showed inhibition of alcohol metabolism by barbiturates. The addition of excess NAD (nicotinamide adenine dinucleotide), which suppresses this inhibition, suggests that the barbiturates do not inhibit liver alcohol dehydrogenase, but, instead, inhibit the metabolic conversion of NADH (reduced form) to NAD.

1363. Trenholm, H. L., Maxwell, W. B., Paul, C. J., Wiberg, G. S., and Coldwell, B. B.
BIOCHEMICAL ASPECTS OF THE INTERACTION OF ETHANOL WITH BARBITURATES.

Canad. J. Biochem. (Ottawa), 48(6): 706-711 (29 ref.),

1970.

E – exp. cont. – exp. comp. – DC (decrease) – mammals – in vitro – other drug lev. – metab. proc. – sed., hypnot. – *CAAAL-0 B-0564.

To determine whether barbiturates interfere with alcohol metabolism, an enzymatic rat liver fraction containing alcohol dehydrogenase (ADH) was used to determine the rates of ADH-catalyzed alcohol oxidation and of acetaldehyde reduction, using ethanol and various concentrations of pentobarbital. With enzymatic activity being dependent on ethanol concentration when pentobarbital was present, results showed little pentobarbital effect on reaction rates in the enzyme blank (no ethanol) or with 0.05 mM ethanol, while enhancement of enzyme activity occurred with 0.5 and 5.0 mM ethanol when

pentobarbital concentration was greater than 1.0 mM. At higher pentobarbital concentrations, ADH stimulation was observed to be directly dependent on the barbiturate concentration, during inhibition of ethanol metabolism by pentobarbital at incubation times greater than 60 min. Results suggested that pentobarbital was stimulating the activity of ADH independently of the direction of the reaction. The elevated acetaldehyde concentrations suggested that pentobarbital was inhibiting ethanol metabolism, not through enzymes involved in acetaldehyde metabolism, but by pentobarbital retardation of NAD regeneration, i.e., inhibition of oxidation of extramitochondrial NADH.

1364. Truitt, E. B., Jr., Duritz, G., Morgan, A. M., and Prouty, R. W.

DISULFIRAMLIKE ACTIONS PRODUCED BY HYPOGLYCEMIC SULFONYLUREA COMPOUNDS.

Quart. J. Stud. Alcohol (New Haven), 23: 197-207 (19 ref.), 1962.
 E – exp. cont. – DC (sensit.) – acute admin. – in vivo – glands – metab. proc. – cardiovasc. agents
 – hormones, hormone antag. – *CAAAL-9685-B2 A-1447.

The alcohol-intolerance reactions produced by tolbutamide and chlorpropamide were investigated in rats, dogs, and cats. The mechanism of the reaction was studied, particularly in order to determine whether it results from the hypoglycemic action of the drugs, and the possibility of an adrenergic blocking action was investigated. Tolbutamide (65-250 mg/kg ip), administered to rats 90 min before 1 g/kg ethanol ip, increased acetaldehyde (AcH) to levels 7.0-13.5 gamma/ml higher than in controls given only ethanol. The vasopressor phase of blood pressure responses in cats after injection of 30 mg/kg 2% AcH was deeper by an average of 16.4 mm Hg; after 200 mg/kg tolbutamide, the vasopressor response to 1 gamma/kg 0.002% epinephrine was unchanged. Disulfiram (100-250 mg/kg po for 3 days) and calcium carbimide (25 mg/kg po, administered 4 hr prior to testing) deepened and prolonged the vasopressor response to AcH and epinephrine, whereas 250 mg/kg tolbutamide iv decreased the vasopressor effect of AcH. Tests with insulin and glucose ruled out a hypoglycemic explanation for the action of AcH vasodilatation, and tests with atropine indicated that bradycardia of vagal origin was not the cause. Cardiovascular changes seem unrelated to adrenergic blocking mechanisms. It is concluded that the hypoglycemic sulfonylurea drugs should be considered as less toxic drugs than disulfiram and calcium carbimide in the treatment of alcoholism.

1365. Tuovinen, P. I.

A CASE OF CARBON TETRACHLORIDE POISONING WITH ANURIA TREATED BY UNILATERAL DISCISSION OF THE RENAL CAPSULE.

Ann. Chir. Gynaec. Fenn. (Helsinki), 38: 169-172 (5 ref.), 1949.
 E – SEC – DC (unspec.) – drug-dep. humans – liver, kidney – anti-infectants – elect., water-bal. agents
 – hormones, hormone antag. – *CAAAL-0 A-1178.

A case of carbon tetrachloride (CCl₄) poisoning is reported, concerning a 57 yr-old alcoholic who had been addicted to alcohol since his thirtieth yr, and who, prior to CCl₄ exposure, had been drunk every week, sometimes for a whole week without interruption. On July 15, 1/2 l of brandy was consumed; on July 16, 1/2 l of eau de vie and 4 “grogs”; and, on July 17, a bottle of hair lotion containing alcohol, perfume, and 3 g CCl₄. This caused the man rapidly increasing pains in the abdomen and decreasing micturition. In a week's time, he developed anuria. At the critical phase, an operation was performed in which the left kidney was removed. The patient achieved fluid balance 5 days after the operation. For 4 weeks, there was exudation smelling of urine from the wound. The patient was released from the hospital as convalescent 5 weeks after the operation.

1366. Uchermann, R.

SÖVNPROBLEMER HOS ALKOHOLIKERE I ABSTINENSFASEN: ERFARINGER MED ET NYTT SOVEMIDDEL: MOGADON. [Sleeping problems in alcoholics during the withdrawal phase: experiences with a new soporific: mogadon].

T. Norsk. Laegeforen. (Oslo), 87(9): 757-760 (3 ref.), 1967.
 N – SEC – general – DC (unchanged) – drug-dep. humans – CNS – analg., antipyret. – autocoids
 – elect., water-bal. agents – sed., hypnot. – tranquilizers – unclass. ther. agents – *CAAAL-0
 B-0473.

500 alcoholics of all categories were treated at the clinic, upon admission, with sodium chloride (5 mg), acetylsalicylic acid (1 g), disulfiram (0.2 g), and antihistamine (0.05 g), followed by valium (10 mg, and then up to 100 mg/day po or im) for appreciable withdrawal symptoms. 10 mg mogadon (nitrazepam) was tested on patients suffering from abstinence symptoms after the initial treatment mentioned above, under controlled conditions. 80 of the 100 patients initially tested slept restfully during the first 3 nights, and 95 the following nights. Mogadon proved effective in patients suffering from nervous symptoms, but had little effect in cases of withdrawal symptoms in chronic alcoholism. The drug proved also satisfactory in ambulatory patients treated for alcoholic abstinence. No habituation, withdrawal syndrome, or potentiation with alcohol were observed in the mogadon tests.

1367. Ukai, M.

KETCHŪ ALKOHOL-RYŌ NO SHŌCHŌ NI OKITE. DAI YON HŌ: CALCIUM-EN EKI NARABI NI [ARUKARI-SEI] RINSAN-EN EKI CHŪNYŪ NO KETCHŪ ALKOHOL-RYŌ NI OYOBOSU EIKYŌ. [The states of the blood alcohol volume. IV. The influence of an injection of calcium salt and alkaline phosphate solutions on the blood alcohol volume]. Okayama Igakkai Zasshi (Okayama), 52: 394-406 (29 ref.), 1940.
 J – GS – exp. cont. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – hormones, hormone antag. – *CAAAL-2741-A2 A-1179.

The alcohol content of the blood and the urine of male rabbits was determined after 10 cc of 10% alcohol sol/kg (po) and: a) after iv injection of 10 cc of 3% calcium chloride sol, and b) after iv injection of alkaline phosphate sol. After calcium chloride, the alcohol concentration in blood and urine was considerably below the control values, and return to normal level was quicker. After the alkaline phosphate sol, there was little change.

1368. Umiker, W., and Pearce, J.

NATURE AND GENESIS OF PULMONARY ALTERATIONS IN CARBON TETRACHLORIDE POISONING.

Arch. Path. (Chicago), 55: 203-217 (10 ref.), 1953.
 E – SEC – general – case hist. – DC (add., infra-add., unspec. incr.) – post-mort. – humans – drug-dep. humans – respir. – anti-infectants – *CAAAL-0 A-1324.

The autopsy protocols and microscopic sections from 26 cases of fatal carbon tetrachloride poisoning were studied with respect to changes in the lung, and the general clinical, roentographic, and pathological features, as they apply to the lung, are described. The patients were all 21-45 yr-old men, of whom 12 were poisoned by ingestion of carbon tetrachloride, and 13 by inhalation. Their courses were characterized by clinical and laboratory evidence of liver and kidney failure, and time of death varied from 1-16 days after exposure, the greatest number of deaths occurring on the tenth day. A history of alcoholism was frequently found, and many first became ill after a bout of acute alcoholism—9 were alcoholics, 1 a heavy drinker, and 4 drank carbon tetrachloride, thinking it to be whiskey or gin.

1369. Ungerleider, J. T.

ALCOHOL, CONVULSIONS AND TRANQUILIZERS: A CLINICAL AND ELECTROENCEPHALOGRAPHIC STUDY.

J. Nerv. Ment. Dis. (Baltimore), 127(6): 518-527 (50 ref.), 1940.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – drug-dep. humans – acute admin. – in vivo

– blood lev. – other drug lev. – cardiovasc. – CNS – respir. – tranquilizers – *CAAAL-8792-D1
A-1180.

30 electroencephalograms were made on a male alcoholic who was given alcohol alone (120 cc absolute alcohol dissolved in fruit juice), or alcohol in combination with: chlorpromazine (100 and 200 mg), promazine (100 and 200 mg), or reserpine (2.5 mg). The patient became hostile after alcohol and the alcohol-tranquilizer combinations; he became drowsy on alcohol plus promazine or chlorpromazine, but not after either tranquilizer alone. Several hr after the reserpine-alcohol combination, he became dyspneic and flushed, and experienced headache—this condition disappeared overnight. None of the EEG tracings were abnormal.

1370. Urechia, C. -I., and Dragomir, L.
INTOXICATION BARBITURIQUE, TRAITÉE PAR DES INJECTIONS
INTRAVEINEUSES D'ALCOOL À 30 P. 100. [Barbiturate intoxication, treated by intravenous
injections of 30% alcohol].
Paris Médical (Paris), 93: 187 (1 ref.), 1934.
F – general – case hist. – DC (antidotal) – drug-dep. humans – cardiovasc. – CNS – senses – skel.,
muscle, skin – analg., antipyret. – barbiturates – *CAAAL-0 A-1181.

A case is reported of an alcoholic who ingested 2 g luminal in a suicide attempt. Upon reaching the hospital, he was found to have a pulse of 72, blood pressure of 15-9, temperature of 37.5°, constipation, headache, insufficient convergence, horizontal bilateral nystagmus, lack of ocular reflexes, and diminished muscular strength; his appearance was that of a drunkard. 20 cc 30% ethanol was given iv, followed by a second injection 2 hr later, after which sleep became less deep and the headache was much relieved. 7 hr after a third dose of 20 cc alcohol, diplopia and hypersomnia disappeared, the man could talk and walk, and his mental state was improved. The next morning, he awoke complaining of constipation and a mild headache, and was given a purgative and a pyramidon tablet. In the evening, the patient was again given an injection of 20 cc alcohol. By the third day, he had completely recovered.

1371. Vachetta, A.
ALKOHOL-CHLOROFORMNARCOSE. [Alcohol-chloroform narcosis].
Berliner Klinische Wochenschrift (Berlin), 20: 11 (0 ref.), 1883.
G – general – DC (add., infra-add., unspec. incr.) – humans – mammals – cardiovasc. – CNS –
anesthetics – *CAAAL-0 A-1182.

Chloroform was administered to human subjects and dogs 1/2 hr prior to alcohol (wine) in a sufficient dose to stimulate heart action. In all cases, this application was more effective than the usual narcosis. The method was employed in 1880 in major surgery. The patient was given 60-100 cc wine 1/2 hr prior to operation. Not all cases showed the same degree of sensitivity, with regard to achieving a rapid and deep narcosis, but none showed any danger symptoms. In conclusion, the author points out the post-operative advantage of the alcohol-chloroform narcosis, and recommends its general employment.

1372. Vaillant, G. E., Brighton, J. R., and McArthur, C.
PHYSICIANS' USE OF MOOD-ALTERING DRUGS: A 20-YEAR FOLLOW-UP REPORT.
New Eng. J. Med. (Boston), 282: 365-370 (14 ref.), 1970.
E – SEC – general – conj. addict. – humans – psychol. perform. – CNS – amphetamines – *CAAAL-0
B-0565.

This is a 20-yr follow-up study on the use of mood-altering drugs and alcohol by physicians. The subjects were 45 physicians and 90 non-physician controls obtained by selection. The data concerning

use of stimulants, tranquilizers, sedatives, alcohol, and tobacco, as well as reports on physical fitness, psychological stability, etc., were obtained by questionnaire and were tabulated. The doctors were found to have fewer problems with alcohol; however, they did take more pharmacological agents affecting the CNS than the matched controls. Also, the heavy conjunctive use of drugs, alcohol, and tobacco occurred. All 3 physicians classed as requiring hospitalization or suffering "clear damage to socioeconomic status owing to use of mood-altering drugs or alcohol" used drugs and alcohol to excess, and others in the next most seriously affected group were also prone to conjunctive abuse. Only 3% of men in the 2 "little drug use" categories smoked heavily (2 packs or more/day); this percentage rose to 12% in the group of men who had trouble controlling drinking, and to 19% in the group of abusive alcohol and drug users. It is concluded that psychological instability in youth positively leads to adult drug use. The results of a few other similar reports are discussed.

1373. Valentino, C.

ALCOOL ET STRYCHNINE; ALCOOL ET VENIN. [Alcohol and strychnine; alcohol and venom].

Presse Med. (Paris), 13: 579-581 (2 ref.),

1905.

F – exp. cont. – DC (decrease) – other org. – acute admin. – in vivo – CNS – stimulants – *CAAAL-0 A-1183.

The author discusses the reaction mechanism of alcohol and strychnine. Experiments were conducted on the antagonism of alcohol and snake venom in chickens. 1 mg venom im and 4 cc 90% alcohol sc, administered simultaneously, induced death in 6 hr. The same doses administered 1 and 2 hr apart caused death 2 and 6 hr after venom, respectively. All chickens survived sc injections of 0.6 mg venom in combination with 4 cc 90% alcohol sc, reviving in 62 hr without any after-effects, as compared to controls which died in 25 hr.

1374. Valicenti, J. F., Jr., and Newman, W. H.

CARDIAC SIZE, CONTRACTILE FORCE AND WALL TENSION FOLLOWING ETHANOL, PENTOBARBITAL AND OUABAIN.

Fed. Proc. (Bethesda), 27(2): 568 (0 ref.),

1968.

E – SEC – abst. – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – cardiovasc. – barbiturates – *CAAAL-0 B-0996.

Simultaneous recordings in open-chest dogs under pentobarbital anesthesia were made from the left ventricle with an isotonic strain arch, a conventional isometric strain gage arch, and an arch designed to record distending wall tension. Aortic flow and arterial pressures were also monitored. Successive, alternated equal-vol iv infusions of ethanol (2 g/kg) and sodium pentobarbital (30 mg/kg) were administered over periods of 25 min. Both drugs produced comparable depressions of contractile force to about 50% of control. The decreased myocardial efficiency in dogs under pentobarbital anesthesia noted by other authors was also found. Ethanol produced greater dilation, and a greater increase in wall tension, with less decrease in arterial pressure. The changes in wall tension can be related to the conditions of the Laplace Principle. Ouabain grossly decreased size and wall tension, while increasing contractile force.

1375. Vapaatalo, H., and Karppanen, H.

COMBINED TOXICITY OF ETHANOL WITH CHLORPROMAZINE, DIAZEPAM, CHLORMETHIAZOLE OR PENTOBARBITAL IN MICE.

Agents and Actions (Birkhauser), 1(2): 43-45 (10 ref.),

1969.

E – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – chronic admin. – in vivo – CNS – respir. – barbiturates – tranquilizers – *CAAAL-0 B-0583.

The combined toxicity of ethanol (E) with chlorpromazine (CPZ), diazepam (D), chlormethiazole (CM), or pentobarbital (Pb) was studied in mice. The mice were given 800 mg/kg ip of 33% E

simultaneously, or 1, 3, 6, 12, or 24 hr before the iv drug injection. Other mice received the same E injection daily for 6 days before drug administration, and a third group received ip administration of 20 mg/kg CPZ, 15 mg/kg D, 170 mg/kg CM, or 70 mg/kg Pb 3 hr before the iv injection of 66% E. Pretreatment with E significantly altered only the drug toxicity of CM, the LD₅₀ decreasing from 340-180 mg/kg. CM was also the only drug pretreatment which significantly decreased the LD₅₀ of alcohol (3150 ± 168-2700 ± 142 mg/kg). The 6-day E treatment significantly modified only the LD₅₀ of Pb (140-115 mg/kg). It is concluded that E plus CM is the most toxic combination in mice in acute experiments, whereas prolonged E treatment increases Pb toxicity the most. CPZ and D seem to be the safest drugs to combine with alcohol.

1376. Vapaatalo, H., and Karppanen, H.

INFLUENCE OF ETHANOL ON THE TOXICITY OF SOME PSYCHOTROPIC DRUGS.

Scand. J. Clin. Lab. Invest. (Oslo), 23: 77 (O ref.), 1969.
E – abst. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo
– CNS – sed., hypnot. – tranquilizers – *CAAAL-0 B-0566.

The influence of ethanol (E) on the iv toxicity of chlorpromazine (CPZ), diazepam (D), chlormethiazole (CM), and sodium pentobarbital (B) was studied in white, male mice. The mice received 0.8 mg/kg E ip, simultaneously or 1, 3, 6, 12 or 24 hr before the iv drug injection. The LD₅₀s of the drugs alone at 24 hr were: CPZ, 46; D, 31; CM, 340; and B, 140 mg/kg. Ethanol lowered the LD₅₀ of CPZ and D only after 24 hr, to 33 and 23 mg/kg, respectively. The LD₅₀ of CM was decreased 3 hr after E injection, and, after 6 hr, was 180 mg/kg. Simultaneous injection of E and B reduced the LD₅₀ of B to 120 mg/kg. Treatment for 6 days with E did not alter the toxicity of CPZ, D, or CM. It is concluded that the toxicity of CPZ and D is not potentiated by E until 24 hr after E administration, whereas the toxicity of CM and B is potentiated when E is given simultaneously or shortly before the drug.

1377. Varney, D. H. M.

SOBERING-UP.

Brit. Med. J. (London), 2: 1025 (1 ref.), 1961.
E – general – DC (antidotal) – humans – CNS – stimulants – *CAAAL-9630-N14 A-1184.

A method of rapidly “sobering-up” a patient brought in comatose after drinking is described. It consists of an injection of 4 ml nikethamide iv, and not too quickly. When giving large doses of nikethamide to this type of patient, it is advisable to have assistance at hand to control a boisterous response. In a case described, a man in a comatose state began to make violent movements as soon as the syringe was withdrawn, and promptly attempted to assault the author.

1378. Vatteteau

OBSERVATIONS RELATIVES AUX EFFETS DE L'AMMONIAQUE ADMINISTRÉE CONTRE L'IVRESSE. [Observations concerning the effects of ammonia administered to counteract intoxication].

Recueil de Mémoires de Médecine, de Chirurgie, et de Pharmacie Militaires (Paris), 23: 311-319 (O ref.), 1827.
F – general – case hist. – DC (antidotal) – humans – CNS – anesthetics – stimulants – *CAAAL-0 A-1185.

A case is cited in which severe alcoholic intoxication was treated with 12 drops of ammonia in 1 oz of sweetened water. 5-6 min after administration of the potion, the patient had calmed down considerably. A second dose of 6 drops of ammonia in 2 oz of lime blossom tea brought about complete tranquility. The patient was asleep 2 hr later, and was able to resume his normal work the next morning. In another case, treatment consisted of 15 drops of ethyl ether in 1 oz of sweetened water.

No change was effected with this dose. A second dose of 12 drops of the substance in 1 oz of lime blossom tea brought some improvement 5-6 min later, and a third dose of 8 drops of ethyl ether in 2 oz of lime blossom tea brought recovery of the intellectual faculties, restful sleep, and resumption of work on the following day. 2 other cases of treatment with ammonia are described.

1379. Veale, W. L., and Myers, R. D.

DECREASE IN ETHANOL INTAKE IN RATS FOLLOWING ADMINISTRATION OF *p*-CHLOROPHENYLALANINE.

Neuropharmacology (Oxford), 9(4): 317-326 (23 ref.),

1970.

E – exp. cont. – exp. comp. – DC (sensit.) – mammals – acute admin. – chronic admin. – in vivo – psychol. perform. – CNS – metab. proc. – *CAAAL-0 B-0997.

Rats were offered water and an ethanol sol in a free-choice situation. The concentration of ethanol was systematically increased from 3% to 30% during repeated 10- or 11-day sequences. 300 mg/kg *p*-chlorophenylalanine (CPA) was administered po on every day of an ethanol preference sequence. The drug was given to 3 groups of ethanol-selecting rats: those which showed an initial predisposition, those which were acclimatized, and those which experienced stress produced by random electric shock after avoidance conditioning. CPA produced a significant reduction in the selection of ethanol by both the predisposed and acclimatized groups, and a further reduction was effected in the sequence after the drug was terminated. In the stress group, CPA exerted its main effect during the period after administration had ceased. CPA inhibits the hydroxylation of tryptophan, and thereby lowers serotonin levels. Explanations offered for the CPA effect on ethanol preference include: cerebral 5-hydroxytryptamine (5-HT) changes, the possibility that 5-HT and ethanol metabolism may be inter-related, or the possibility of reduced pain threshold and a shift in sensitivity to sensory stimulation, so that the usual aversion to the taste of alcohol is intensified. Long-term metabolic changes may also be involved.

1380. Veldstra, H.

SYNERGISM AND POTENTIATION: WITH SPECIAL REFERENCE TO THE COMBINATION OF STRUCTURAL ANALOGUES.

Pharmacol. Rev. (Baltimore), 8: 339-387 (366 ref.),

1956.

E – SEC – general – review – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – humans – absorp., distrib., stor. – CNS – barbiturates – *CAAAL-0 A-1186.

The author makes an extensive review of the literature, and discusses the question of synergism and potentiation in considerable detail. Attempting to end the confusion over terminology, he notes that the term "synergism" is sufficient—since "potentiation" means to "endow with power", and since, in synergistic drug combinations, the components possess a power which is not altered but enhanced in effectiveness, no real potentiation occurs, and usage of the term should be dropped. The problem of ethanol interaction is briefly mentioned with respect to barbiturates and octyl alcohol.

1381. Vendsborg, P. B., and Schambye, P.

THE INFLUENCE OF 2,4-DINITROPHENOL ON METABOLIC CHANGES CAUSED BY ETHANOL IN THE PERFUSED RAT LIVER.

Acta Pharmacol. (Copenhagen), 28(2): 113-123 (28 ref.),

1970.

E – SEC – exp. cont. – DC (unspec.) – mammals – acute admin. – in vitro – absorp., distrib., stor. – liver, kidney – metab. proc. – stimulants – *CAAAL-15149 B-1022.

The mechanism by which ethanol inhibits the tricarboxylic acid cycle was investigated in controlled experiments on isolated rat livers perfused with human erythrocytes. After a 45-min equilibration, samples of the perfusion medium were taken, to determine the lactate/pyruvate (L/P) and hydroxybutyrate/acetoacetate (H/A) ratios (as parameters of extra- and intramitochondrial NADH/NAD

ratios, respectively), and 3 ml (1 M) ethanol was added. 30 min later, samples were again taken, and 1 ml (1 M) ethanol and 4 ml (25 mM) dinitrophenol (DNP) were added. After another 30 min, the last samples were taken. The oxygen uptake and carbon dioxide production were recorded as well. Ethanol slightly increased the L/P ratio, while DNP effected a significant increase. Also, ethanol induced a 3-fold rise in the H/A ratio; this was completely abolished by DNP. Oxygen uptake was unaffected by ethanol, but was increased by 20% after DNP. Carbon dioxide production was decreased by 54% after ethanol, while DNP caused a 74% increase. The results indicate that extra- and intramitochondrial NADH/NAD ratios are independent of each other, and correlate with the TCA cycle. Ethanol directly inhibits the extramitochondrial NADH/NAD ratio, causing an indirect inhibition of the intramitochondrial ratio, and a simultaneous decrease in carbon dioxide production.

1382. Venho, I., Eerola, R., Venho, E. V., and Vartiainen, O.
 SENSITISATION TO MORPHINE BY EXPERIMENTALLY INDUCED ALCOHOLISM IN WHITE MICE.
 Ann. Med. Exp. Biol. Fenn. (Helsinki), 33: 249-252 (1 ref.), 1955.
 E – exp. cont. – cross-tol. – mammals – acute admin. – chronic admin. – in vivo – dose resp. – CNS – analg., antipyret. – *CAAAL-1508-D2 A-1187.

Increasing doses of alcohol were administered to 30 female white mice for 3 1/2 months. The alcohol sol started with 0.5%, and was gradually increased to 10% for the last six weeks. At the end of the 3 1/2-month period, the alcohol was stopped for 1 day to allow the blood alcohol to decline to normal levels, and then morphine (0.14, 0.18, and 0.40 mg/g—corresponding, in normal mice, to the LD₅, LD₁₀, and LD₅₀, respectively) was given sc. The degree of sensitization was higher than expected. All mice which received an LD₅₀ dose calculated for normal mice were killed, and it is considered highly probable that the LD₃₀ dose, calculated on normal mice, would have been sufficient to cause 100% mortality in the alcohol-habituated group.

1383. Verron, G.
 VERGLEICHENDE UNTERSUCHUNGEN ÜBER DEN SORBITSTOFFWECHSEL MIT UND OHNE ALKOHOLZUSATZ. [Comparative investigations on sorbit metabolism, with and without alcohol].
 Z. Ges. Inn. Med. (Leipzig), 20(9): 278-283 (39 ref.), 1965.
 G – ES – RS – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – metab. proc. – elect., water-bal. agents – *CAAAL-11569-B1 B-0474.

14 children with nutritional difficulties received alcohol (0.2 mg 96% alcohol/kg) plus sorbit (0.5 g/kg in 20% sol iv). It was found that the ethanol delayed the breakdown of sorbit, as shown by comparison of the curves representing sorbit concentration in the blood, with and without ethanol. The result was confirmed by the course of the curves representing the concentration of fructose and glucose in the blood. For the first 45 min after the administration of sorbit and ethanol, the level of fructose in the blood was materially lower than after an injection of sorbit alone. After this period of time, the fructose values lay above the comparative curve, as the protracted breakdown of sorbit stipulated a longer fructose retention. It is also possible that the retardation of the breakdown of fructose by ethanol contributes to the latter result.

1384. Videla, L., and Israel, Y.
 FACTORS THAT MODIFY THE METABOLISM OF ETHANOL IN RAT LIVER AND ADAPTIVE CHANGES PRODUCED BY ITS CHRONIC ADMINISTRATION.
 Biochem. J. (London), 118: 275-281 (48 ref.), 1970.
 E – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – in vitro – other drug lev. – liver, kidney – metab. proc. – nutritive agents – *CAAAL-0 B-0584.

The effects of uncoupling agents (substances which increase the mitochondrial oxidation rate) on ethanol metabolism in rat liver slices, and of chronic ethanol administration upon ethanol catabolism *in vivo*, were studied. The rats treated chronically with alcohol were fed *ad libitum* a protein-enriched diet, to prevent triglyceride accumulation in the liver. Cyanide was found to inhibit ethanol metabolism (EM): 2,4-dinitrophenol increased it 160%, but, in a 95% oxygen atmosphere, it had no effect (the metabolism having been already 100% increased by the oxygen). Arsenate increased EM by 80% in a concentration of 10 mM, but inhibited it in a 37.5 and 50 mM concentration. Fructose also increased EM, as did chronic alcohol administration. Dinitrophenol had no effect on the already elevated metabolism of the liver slices from the ethanol-treated rats. Dinitrophenol increased the lactate and pyruvate production in both ethanol-treated and other rats, but did not affect the lactate-pyruvate ratio. The chronic ethanol treatment did not effect alcohol dehydrogenase activity, but did increase succinate dehydrogenase activity by 40%. Among other conclusions, the author suggests that the re-oxidation of the co-enzyme, NADH, is the limiting step in the metabolism of ethanol.

1385. Villiaumey, M. J.

A PROPOS D'UN CAS D'INTOLÉRANCE AUX BOISSONS ALCOOLISÉES AU COURS D'UN TRAITEMENT PAR UN ANTIFONGIQUE DE SYNTHÈSE (DISULFURÉ DE TÉTRA-MÉTHYL-THIURAM). [A case of intolerance to alcoholic beverages during a treatment with a synthetic fungicide (tetramethylthiuram-disulphide)].

Bull. Soc. Franc. Derm. Syph. (Paris), 61: 42 (0 ref.),

1954.

F – general – case hist. – DC (sensit.) – humans – nerv. syst. – respir. – senses – skel., muscle, skin – miscellaneous – unclass. ther. agents – *CAAAL-0

A-1325.

The similarity between the fungicide tetramethylthiuramdisulfide and antabuse (tetraethylthiuramdisulfide), the latter being used in alcoholism therapy, is pointed out, and a case history is cited. A 31 yr-old male applied tetramethylthiuramdisulfide ointment and powder daily as treatment for erythrasma. On the third day, after a meal with wine, his face became flushed and burning hot. In 48 hr, these symptoms became worse—skin temperature rose, and there were throat and lung irritations; coughing; puffiness of the face, neck, and back of hands; ear-aches; and general anxiety. After 3 days, all symptoms ceased and the erythrasma treatment was resumed, but with it the symptoms returned. Abstinence from all alcohol, however, brought about the disappearance of the symptoms, and daily medication was resumed under this condition. The author concludes that tetramethylthiuramdisulfide has similar effects to antabuse in combination with alcohol, and that during, and for some days after, treatment with it, no alcohol should be taken.

1386. Vincenzi, L., Meldolesi, J., Morini, M. T., and Bassan, P.

PROTECTIVE EFFECT OF PHENOBARBITAL AND SKF 525A ON THE ACUTE ETHANOL-INDUCED FATTY LIVER.

Biochem. Pharmacol. (New York), 16(12): 2431-2432 (19 ref.),

1967.

E – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – *in vivo* – liver, kidney – metab. proc. – barbiturates – *CAAAL-0

B-0475.

Experiments were conducted to study whether the experimental modification of microsomal drug-metabolizing enzyme activity influences the acute ethanol-induced fatty liver. Rats were first treated with either phenobarbital (80 mg/kg *ip* 2 hr prior to oral intubation with either glucose or ethanol) or with SKF 525A (80 mg/kg 2 hr prior to oral intubation). The level of hepatic triglyceride in the various experimental groups was recorded. Both phenobarbital and SKF 525A had no detectable effect on the controls, but afforded a clear protection in the ethanol-treated rats. It is hypothesized that the protection observed may be due to a partial inhibition of the pro-oxidative effect of ethanol on the hepatic microsomes, and, from this point of view, the protective mechanism of phenobarbital and SKF 525A seems to be similar to that of antioxidants.

1387. Vlk, H.

ZUR FRAGE DER WECKWIRKUNG DES CORAMIN BEI ALKOHOLVERGIFTUNGEN.

[Stimulating effect of coramine in alcohol poisoning].

Wien. Med. Wschr. (Vienna), 90: 687 (3 ref.),

1940.

G – general – case hist. – DC (antidotal) – humans – mot. perform. – cardiovasc. – CNS – metab. proc. – respir. – *CAAAL-0 A-1326.

Writings on the effects of coramine in alcohol poisoning show noteworthy unanimity, and experimental evidence attributes its arousing effect to its ability to accelerate alcohol oxidation. 2 cases of alcohol poisoning treated with coramine are presented. The first, a 30 yr-old man, was taken unconscious to hospital in an ambulance, showing symptoms of slowly-reacting pupils, superficial breathing, cyanosis, and faint pulse. Usual methods of arousal failed, and 5 cc of coramine were given iv. After only 2 cc had been administered, the patient breathed in deeply and attempted to roll onto his side. The remaining 3 cc were quickly administered, and, before the needle was removed, the patient awoke, jumped off the stretcher, and wanted to leave the examination room. The second case concerned a woman who had drunk about a half a liter of bad brandy (Fuselbranntwein). She was unconscious, had a blue-red face, large pupils, no reflexes, fluttering pulse, and extremely shallow breathing. Within a 10 min period, 15 cc coramine was administered. Improved circulation and breathing followed. The author recommends oral doses in less extreme cases, largely because of the fact that it is difficult to give an injection to an inebriated individual.

1388. Vogel, G.

ÜBER EINE BISHER UNBEKANNTE BIOLOGISCHE WIRKUNG VON

BIER—STEIGERUNG DER PRODUKTION VON LYMPHE. [A hitherto unknown biological action of beer—increase of the production of lymph].

Brauwissenschaft (Nuremberg), 19(8): 307-310 (1 ref.),

1966.

G – ES – FS – exp. cont. – exp. comp. – congen. stud. – mammals – acute admin. – in vivo – blood lev. – other drug lev. – blood comp., sites, lymph – G.I. tract – *CAAAL-12416-B2 B-1023.

On the basis of a chance observation that lymph fluid in the thoracic duct of rats is increased after injection of 10 ml of 20 vol% ethanol/kg into the intestine, the question was investigated whether commercial alcoholic beverages—export and bock beer (40 ml/kg), champagne (30 ml/kg), and cognac and brandy (8 ml/kg)—administered by tube into the duodenum of white, anesthetized rats would have the same effect, and, if so, whether this is due to alcoholic content or to other agents. Control rats received a 4 vol% aqueous ethanol sol or water. Lymph was drawn from the thoracic duct and sampled 1 hr before, and 1 and 2 hr after administration. All test sol, except beer, increased the lymph flow in proportion to their ethanol content. Beer, however, had a stronger effect, and it is concluded that this beverage contains other lymph-stimulating agents in addition to ethanol; these agents must increase the absorption and transport of fats from the intestine. In addition, it is noted that beer is capable of increasing lymphatic transport of drugs from the intestine. Further research is needed to identify this special component of beer. The diuretic action of beer was also investigated, and it was determined that this effect is due to the ethanol content.

1389. Vogel, G., Lehmann, G., Meyering, E., and Wendt, B.

TIEREXPERIMENTELLE UNTERSUCHUNGEN ZUR LYMPHAGOGEN, DIURETISCHEN UND CHOLERETISCHEN WIRKUNG VERSCHIEDENER

ALKOHOLICA. [Experimental investigations of the lymphagogous, diuretic, and choleretic activities of different alcoholic beverages in animals].

Arzneimittelforschung (Aulendorf), 16: 673-677 (7 ref.),

1967.

G – ES – exp. cont. – congen. stud. – DC (unspec.) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – blood comp., sites, lymph – G.I. tract – liver, kidney – metab. proc. – analg., antipyret. – *CAAAL-12416-B2 B-1024.

The lymphagogous effect, on the thoracic duct of anesthetized male and female rats (115-350 g), of po or intraduodenal administration of the following beverages was studied: beer and bock beer (40 ml/kg), champagne (30 ml/kg), cognac and brandy (8 ml/kg), and corresponding amounts of 5.1, 10.1, and 40 vol% aqueous ethanol sol. Lymph samples were taken during 2 1-hr intervals; the rates of bile secretion and urine excretion were also measured. All alcoholic beverages, except beer, increased the production of lymph in proportion to their alcoholic content. Beer was found to possess additional lymphagogous properties in addition to alcohol. As well, it was found that beer has no specific diuretic or choleretic effect in rats, and that beer increases lymphatic transport of quinine (administered into the rat duodenum) by a rise in the quinine concentration of the lymph. It is suggested that the superior lymphagogous effect of beer might be due to an increased production of liver lymph. Beer consumption is recommended in cases where an increased lymphatic transport of macromolecules or corpuscles is desirable.

1390. Voith, K., and Herr, F.

PSYCHOPHARMACOLOGICAL EVALUATION OF A NEW ANTIDEPRESSANT: BUTRIPTYLINE.

Arch. Int. Pharmacodyn. (Gand), 182(2): 318-331 (27 ref.), 1969.
 E – SEC – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
 – in vivo – mot. perform. – CNS – antidepressants – *CAAAL-0 B-0566.

The action of butriptyline was evaluated with respect to the following effects: toxicity; potentiation of a subnarcotic dose of ethanol; potentiation of hexobarbital narcosis; reversal of reserpine-induced hyperthermia; antagonism of tremorine-induced hypothermia, tremor, salivation, and lacrimation; potentiation of amphetamine-induced hyperthermia; runway test; conditioned-avoidance response; ataxia; and anti-spasmodic activity in vitro. 30 min after an ip injection of graded doses of butriptyline and imipramine, 4 g/kg ethanol as a 20% sol was injected ip into mice. The loss of the righting reflex (LRR) was determined, using an "all or nothing" criterion. In non-pretreated mice, the ethanol caused a LRR in a maximum of 5% of the animals, whereas the LRR was universal in pretreated mice. The ED₅₀ for butriptyline was 25 mg/kg, and for imipramine it was 41 mg/kg.

1391. Vollmer, H.

VERSUCHE ÜBER DIE GIFTEMPFINDLICHKEIT WEISSER MÄUSE NACH VORBEHANDLUNG MIT CASEOSAN, SUFROGAL UND ALKOHOL. [Experiments on

the toxophilic condition of white mice after pretreatment with caseosan, sufrogel, and alcohol].
 Naunyn-Schmiedebergs Archiv für Experimentelle Pathologie und Pharmakologie (Berlin), 155: 160-184 (28 ref.), 1930.

G – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged)
 – mammals – acute admin. – chronic admin. – in vivo – dose resp. – CNS – analg., antipyret. – unclass.
 ther. agents – *CAAAL-0 A-1463.

In a control experiment, the onset, duration, and degree of narcosis were recorded after sc administration to white mice of 4.73 mg/g alcohol in a 10% sol containing 0.9% sodium chloride. The same alcohol dose was used, and the same tests applied, in all subsequent experiments. In experiments with caseosan (CA—a casein preparation), various acute and chronic dosages of CA were administered sc to mice, followed by sc injections of alcohol, morphine, quinine, hydroquinone, and colchicine. In another experiment, 0.4 mg/g morphine was administered sc to mice, 1-6 days after injection of alcohol. In a 4th experiment on mice, various doses of sufrogel (a therapeutic colloidal sulphur preparation) were given sc for 3 days, followed by alcohol on the 5th day. In most CA experiments, acute or chronic administration of CA significantly decreased or provided protection against the effect of alcohol, but CA had no effect on sensitivity to the other drugs administered. The effect of CA on alcohol narcosis is probably due to a CA-induced increase in the rate of alcohol oxidation, and is independent of temperature fluctuations. Alcohol pretreatment increased the effect of morphine up

to the 3rd day after alcohol, but had no effect on days 4, 5, and 6. Sufrogel caused fluctuations in body temperature, but sensitivity to alcohol was not influenced.

1392. Volovik, V. M.

O TOLERANTNOSTI ALKOGOLIKOV K BARBITURATAM. [On the tolerance of alcoholics to barbiturates].

Nauchno-issledovatel'skii Psikhonevrologicheskii Institut imeni V.M. Bekhtereva, Trudy (Leningrad), 36: 151-157 (10 ref.), 1967.

R – exp. cont. – cross-tol. – drug-dep. humans – acute admin. – chronic admin. – in vivo – CNS – barbiturates – *CAAAL-0 B-0476.

Investigations were carried out to determine the sedation threshold of alcoholics. 55 chronic alcoholics were used, 37 in their second stage of alcoholism, and 18 in the final stages. 15 healthy subjects made up the control. Injections of 5% sodium amytal showed some increase in the threshold value for secondary alcoholics (5.05 mg/kg tolerance). Terminal alcoholics showed a value of 4.5 mg/kg, and the control group 4.59 mg/kg. Second-degree alcoholics with cerebral malfunctions showed a value of 3.8 mg/kg, and terminal alcoholics with cerebral failings, 3.5 mg/kg.

1393. Von Hagen, D. S.

INTERACTIONS OF CALCIUM ION AND ETHANOL IN SMOOTH MUSCLE FUNCTION.

Ph.D. Thesis, Graduate School of Vanderbilt University, Nashville, Tennessee, U.S.A., 123 pp. (62 ref.), 1965.

E – exp. cont. – DC (decrease) – mammals – in vitro – acid-base, blood pH, elect. – metab. proc. – skel., muscle, skin – elect., water-bal. agents – *CAAAL-0 B-0319.

In this study, 2 parameters of smooth muscle (guinea pig ileum) function were measured—isotonic concentration, and the increase in potassium efflux (produced by acetylcholine or by a medium containing a high concentration of potassium ion). The changes in function produced by varying the external calcium concentration and the ethanol concentration were determined. It was found that the effects of ethanol on contraction induced by acetylcholine and the depolarizing sol, and on the increase in potassium efflux induced by acetylcholine, exhibit progressive changes with increasing concentrations of ethanol, and are markedly influenced by the external calcium concentration. On the other hand, the effect of ethanol on the increase in potassium efflux induced by the depolarizing sol does not exhibit progressive changes with the ethanol concentration, and is not greatly influenced by the external calcium concentration.

1394. Votava, Z., and Dyntarová, H.

COMPARISON OF THE CLOMETHIAZOLE AND DIAZEPAM EFFECTS ON THE BY ALCOHOL INDUCED CHANGES OF EEG AND BEHAVIOR OF RATS.

In: *Collegium Internationale Neuro-Psychopharmacologium (C.I.N.P.). Abstracts. II.* International Conference, Prague, Czechoslovakia, August 11-15, 1970. Prague: C.I.N.P.,

p. 489 (0 ref.) @ 1970.

E – abst. – exp. comp. – presentation – DC (decrease) – mammals – acute admin. – in vivo – mot. perform. – psychol. perform. – CNS – tranquilizers – *CAAAL-0 B-0998.

Brain electrodes were implanted in rats, and electroencephalograph (EEG) recordings were made. 2 g/kg ethanol ip evoked EEG activation, followed by increased motility. 20 mg/kg clomethiazole ip, administered 10 min after the above ethanol dose, decreased motility, but did not affect ethanol-induced EEG activation. 1 mg/kg diazepam ip normalized both the motility and the EEG. It is concluded that clomethiazole differs from minor tranquilizers and barbiturates in its calming effect on alcohol arousal.

1395. Wacker, W. E. C., Haynes, H., Druyan, R., Fisher, W., and Coleman, J. E.
TREATMENT OF ETHYLENE GLYCOL POISONING WITH ETHYL ALCOHOL.
J.A.M.A. (Chicago), 194: 1231-1233 (15 ref.), 1965.
E – general – case hist. – DC (antidotal) – humans – cardiovasc. – CNS – liver, kidney – metab. proc.
– respir. – indust. intox. – *CAAAL-11353-A1 B-0477.

The use of iv ethanol in the treatment of ethylene glycol poisoning is advocated, because ethanol prevents the oxidation of ethylene glycol into oxalic acid, which is toxic to the kidneys. The first 2 clinical cases in which iv ethanol was used successfully are reported. Ethanol therapy for ethylene glycol poisoning should be continued for 3 to 5 days if urine output is maintained; this therapy should supplement supportive measures, including acid-base therapy and dialysis.

1396. Wade, D. J., and Eade, N. R.
THE EFFECT OF PHENELZINE ON THE DEGRADATION OF ETHANOL.
In: Walker, E.G., ed. *Canadian Federation of Biological Societies, Proceedings. II.* Eleventh Annual Meeting, Kingston, Ontario, Canada, June 12-14, p. 41 (0 ref.), 1968.
E – abst. – presentation – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin.
– in vivo – dose resp. – metab. proc. – enzymes – *CAAAL-0 B-0478.

The effect of phenelzine sulphate on ethanol-treated mice was determined. Male Swiss mice were given phenelzine sulphate (40 mg base/kg ip), followed 30 min later by 3.5 g/kg ethanol ip. Phenelzine pretreatment increased the ethanol sleeping time by 300%, and increased the LD₅₀ from a control value of 4.14 g/kg to 5.16 g/kg. Determinations of the ethanol concentrations in whole mice over 1 4-hr period indicated that phenelzine caused a 43% increase in the time required to eliminate 1/2 of the amount of ethanol originally injected.

1397. Wagner, H. -J.
EINFLUSS VON MEDIKAMENTEN AUF DEN ACETALDEHYDSPIEGEL IM BLUT
NACH ALKOHOLZUFUHR (ENZYMATISCHE BESTIMMUNG DES ACETALDEHYD).
[Effect of drugs on the acetaldehyde concentration in the blood after alcohol administration
(enzymatic determination of acetaldehyde)].
Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 46: 70-78 (26 ref.), 1957.
G – exp. comp. – DC (sensit.) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – analg.,
antipyret. – anti-infectants – barbiturates – unclass. ther. agents – *CAAAL-8522-B2 A-1188.

Male rats in groups of 10 received doses of disulfiram (200 to 250 mg/kg), phenylbutazone (0.5 to 1.0 cc/kg), irgapyrin (0.7 cc/kg), phenobarbital (0.25 to 0.75 mg/kg), isoniazid (10 to 20 mg/kg), or phenacetin (50 to 150 mg/kg). Between 1 and 7 hr later, they received 1.5 g of alcohol/kg, which produced a blood alcohol level of 1-1.5°/oo. Blood was analyzed for acetaldehyde, and concentrations of 0.0 mg%-0.3 mg% were found. The previously reported findings of concentrations of 2.0% could not be confirmed.

1398. Wagner, H. -J.
BEEINFLUSSUNG DES INTERMEDIÄREN STOFFWECHSELS DURCH
VERSCHIEDENE MEDIKAMENTE UND NACHFOLGENDE ALKOHOLGABEN. [Effect
of various drugs followed by alcohol on the intermediary metabolism].
Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 46: 575-582 (21 ref.), 1957.
G – exp. cont. – exp. comp. – DC (sensit.) – mammals – acute admin. – in vivo – blood lev. – metab.
proc. – analg., antipyret. – anti-infectants – barbiturates – unclass. ther. agents – *CAAAL-8522-B2
A-1189.

Investigated was the extent of the pyruvic acid and acetaldehyde formation in the serum of rats which were pretreated with antabuse, butazolidin, irgapyrin, luminal, rimifon, or phenacetin, and then given

alcohol, 1.5 g/kg in 20% sol by stomach tube (the enzymatic method was used); the blood alcohol level rose to 1-1.5°/oo. Half of the subjects received drugs but no alcohol, and were used as controls. The pyruvic acid level in the serum of untreated rats was 1.2 ± 0.15 mg%. After 1 to 3 days treatment with 1 of the drugs plus alcohol, the pyruvic acid content rose to 3-5 mg%, and later up to 12 mg%. The acetaldehyde formation never exceeded 0.3 mg%.

1399. Wagner, H. -J.

TÖDLICHE VERGIFTUNG NACH SUBLETALEN ALKOHOL- UND

POLAMIDONGABEN. [Fatal poisoning after sublethal doses of alcohol and polamidone].

Arch. Toxik. (Berlin), 17: 159-164 (6 ref.),

1958.

G – exp. – general – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – dose resp. – blood lev. – other drug lev. – respir. – analg., antipyret. – *CAAAL-8910-D1 A-1190.

Reported is the case of a male polamidone addict, 43 yr old, who died after alcohol ingestion, following an injection of (a later estimated) 32.55 mg of polamidone. The alcohol concentrations in blood and stomach were 1.25 and 4.57°/oo, respectively. The cause of death was probably respiratory failure. Laboratory data are discussed in detail. A potentiating effect of simultaneous alcohol and polamidone ingestion is very likely.

1400. Wagner, H. -J.

DIE MEDIKAMENTÖSE BEEINFLUSSUNG DER LEISTUNGSFÄHIGKEIT UND IHRE BEDEUTUNG FÜR DIE VERKEHRSSICHERHEIT. [Influence of drugs on performance and its significance to traffic safety].

Munch. Med. Wschr. (Munich), 101(7): 275-282 (40 ref.),

1959.

G – SEC – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – CNS – analg., antipyret. – enzymes – gastrointest. agents – sed., hypnot. – stimulants – tranquilizers – *CAAAL-0 A-0046.

Various drugs are discussed, with respect to their dangerous effect on driving. With the exceptions of Vitamins B₁ and A, none of the compounds reviewed has a true performance-increasing effect, because such an effect is only present if the drug adds to available strength, rather than using up the last physical reserves. Drugs reviewed include: antidepressants, stimulant amines, analgesics, barbiturates and other sedatives, tranquilizers, and iproniazid. It is pointed out that some of the drugs discussed increase the depressive effects of alcohol so considerably that abnormal reactions occur, and the driving ability is seriously impaired.

1401. Wagner, H. -J.

DIE BEDEUTUNG DER UNTERSUCHUNG VON BLUT- BZW. HARNPROBEN AUF ARZNEIMITTEL NACH VERKEHR SUNFÄLLEN AUF GRUND DER ÜBERPRÜFUNG VON 2060 PERSONEN. [Significance of blood and urine tests for drugs after traffic accidents on the basis of the examination of 2060 persons].

Arzneimittelforschung (Aulendorf), 11: 992-995 (11 ref.),

1962.

G – ES – SEC – stat. surv. – DC (add., infra-add., unspec. incr.) – mot. vehic. – blood lev. – other drug. lev. – CNS – analg., antipyret. – antidepressants – anti-infectants – miscellaneous – sed., hypnot. – stimulants – *CAAAL-0 A-1191.

2060 more or less alcoholized traffic offenders were medically examined and questioned for drugs taken within 24 hr preceding the accident. 11% confirmed drug use. The percentage increased with age—15.9% for the age group 40-60, and 19.1% in those over 60. Of the drugs, 50% were mild analgesics, 12% gastro-intestinal drugs, 9.6% sedatives, 7% cardiac and circulatory drugs, and the rest various other drugs. Among the sedative users, the percentage of accidents increased by 77%. The findings showed that potentiating effects of drugs on alcohol action were frequently observed.

1402. Wagner, H. -J.

ARZNEIMITTEL UND VERKEHRSSICHERHEIT. [Drugs and traffic safety].

Therapiewoche (Karlsruhe), 12: 291-297 (22 ref.),

1962.

G – SEC – general – DC (add., infra-add., unspec. incr.) – humans – CNS – metab. proc. – anti-infectants – sed., hypnot. – *CAAAL-0 A-1192.

In a general discussion, it is pointed out that even minute quantities of alcohol and sedatives or hypnotics (but also many other types of drugs) may have a potentiating effect, with severe consequences for the motorist. If the interaction between alcohol and the drugs results in synergism, then the development of pathological and toxic metabolites will take place in a manner similar to the alcohol-antabuse reaction. The author asks the pharmaceutical firms to use labels indicating the danger of synergism between alcohol and drugs (e.g., sedatives, hypnotics, or of psychopharmacological agents).

1403. Wagner, H. -J.

VERGLEICHENDE UNTERSUCHUNGEN ÜBER DIE ZAHLENMÄSSIGE BEZIEHUNG ZWISCHEN MEDIKAMENT- BZW. ALKOHOLBEEINFLUSSTEN

VERKEHRSTEILNEHMERN. [Comparative studies of the quantitative relationship between drivers under the influence of drugs and alcohol].

Zentralblatt für Verkehrs-Medizin, Verkehrs-Psychologie, Luft- und Raumfahrt-Medizin (Munich), 9(3): 1-4 (12 ref.),

1963.

G – SEC – stat. surv. – DC (add., infra-add., unspec. incr.) – mot. vehic. – humans – CNS – sed., hypnot. – *CAAAL-0 A-0012.

The roles of alcohol and drugs in traffic accidents, and the incidence of use in males and females and in various age groups, are reported for 2 German cities. Evaluation of 10,000 case histories of drivers involved in accidents while under the influence of alcohol showed that 10.3%-14.5% had also taken drugs during the preceding 24 hr. Sedatives and hypnotics were found to influence driving ability most. Surveys of the frequency of traffic accidents connected with drug and alcohol ingestion are analyzed.

1404. Wagner, H. -J.

ALKOHOL, PSYCHOPHARMAKA UND VERKEHRSSICHERHEIT. [Alcohol, psychopharmacological agents, and traffic safety].

Monatskurse für die Ärztliche Fortbildung (Munich), 13(8): 454-457 (0 ref.),

1963.

G – SEC – general – DC (add., infra-add., unspec. incr.) – mot. vehic. – CNS – metab. proc. – barbiturates – hallucinogens – sed., hypnot. – stimulants – *CAAAL-0 A-0050.

Analgesics, barbiturates, sedatives, hypnotics, neuroleptics, tranquilizers, and stimulants, used alone and in combination with alcohol, are discussed in relation to traffic safety. Medical questionnaires showed that about 10-12% of drivers involved in accidents had taken drugs in the previous 24 hr. The possible synergistic effect of drug-alcohol combinations is pointed out. The synergistic effects may be caused by toxic substances formed through enzyme blockage, or by action of both compounds on the same CNS functions.

1405. Wagner, H. -J., and Schmitz, B.

ÜBER DEN EINFLUSS VERSCHIEDENER GRUPPEN VON PHARMAKA AUF DIE ALKOHOL-OXYDATION. [Concerning the influence of different groups of drugs on alcohol oxidation].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 57: 240-249 (9 ref.),

1966.

G – ES – exp. cont. – exp. comp. – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – autocoids – cardiovasc. agents – stimulants – tranquilizers – *CAAAL-0 B-0479.

The influence of 10 drugs (analgetics, antihistamines, drugs acting on the blood vessels, antihypertensives, neuroleptics, and drugs which inhibit the appetite) on the blood alcohol curve was tested on rabbits. In 1 experiment, 0.8 g/kg of 30% alcohol iv was given 60-70 min after the drugs (in doses equivalent to the daily max dose for humans). Compared with the controls (alcohol alone), no statistically significant difference in the blood alcohol curves was found.

1406. Wagner, H. -J.

ABGRENZUNG DER KLINISCHEN ZUSTANDSBILDER, DIE INFOLGE EINER ALKOHOL-ARZNEIMITTEL KOMBINATIONSWIRKUNG AUFTRETEN. [Definition of the clinical state which appears as a result of an alcohol-drug interaction].

In: *Alkohol und Verkehrssicherheit*: Konferenzbericht der 5. Internationalen Konferenz über Alkohol und Verkehrssicherheit. [Alcohol and traffic safety: proceedings of the 5th International Conference on Alcohol and Traffic Safety]. Freiburg im Breisgau, West Germany, 1969. Freiburg im Breisgau: Hans Ferdinand Schulz Verlag, pp. VI.39-VI.46 (0 ref.) @ 1969.

G – general – presentation – DC (antidotal) – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – mot. vehic. – humans – drug-dep. humans – blood lev. – mot. perform. – psychol. perform. – cardiovasc. – CNS – metab. proc. – anesthetics – antidepressants – barbiturates – hormones, hormone antag. – nutritive agents – sed., hypnot. – tranquilizers – *CAAAL-0 B-1025.

The importance and implications of alcohol-drug interactions in traffic medicine are discussed, and the various types of combined effects are outlined. 4 major types of interaction are distinguished: (a) alteration of the alcohol effect, (b) alteration of the drug effect, (c) production of new effects, and (d) mixed forms of the other reaction types. The alcohol effect can either be weakened—by stimulants, for example, so as to induce a false sobriety—or strengthened—by various sedatives and hypnotics, such as morphine derivatives, neuroleptics, tranquilizers, antidepressants, etc. The individual drug effect can also be weakened, as is seen when chronic alcohol abuse leads to a shortening of barbiturate narcosis, or strengthened, since the increase of the alcohol effect by a given drug is usually accompanied by an increase in the effects of that drug, due to an alteration in drug metabolism. By production of new effects, the author understands side effects—flushed face, headache, fatigue, increased pulse, fall in blood pressure, etc.—which occur, for example, after alcohol ingestion and concomitant cyanamide exposure or antabuse intake. It is concluded that, although the range of metabolic reactions and the total of possible combinations of alcohol and drugs are limited, a larger variety of resulting interaction effects is possible.

1407. Wagner, K., and Wagner, H. -J.

NIL NOCERE! GEFAHREN EINER MEDIKAMENTÖSEN BEHANDLUNG VON ALKOHOLBEEINFLUSSTEN UNFALLVERLETZTEN (MIT BARBITURATEN, MORPHIN UND POLAMIDON). [Nil nocere! Hazards of treatment of accident victims who are under the influence of alcohol (with barbiturates, morphine and polamidone)].

Munchen. Med. Wschr. (Munich), 100(49): 1923-1925 (15 ref.), 1958.

G – exp. cont. – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – dose resp. – blood lev. – CNS – respir. – analg., antipyret. – *CAAAL-8910-D1 A-1193.

The author points to the dangers of administering barbiturates or morphine to persons injured in traffic whose prior alcohol consumption cannot be determined. Animal experiments are reported which, depending on the dose combination, show that either an additive or a potentiating synergism exists between alcohol and polamidone. 315 mice were used, and the LD₅₀ was established for 95% ethanol at 8442 ± 666 mg/kg, and for polamidone at 54.5 ± 6.5 mg/kg. After application of 11.5 mg polamidone and various alcohol doses, a potentiating effect occurred. In a reported case, a man was found dead after an injection of 33 mg polamidone, having had an alcohol blood level of 1.26°/oo. Physicians treating emergency cases are warned about this possibility of lethal potentiation.

1408. Wagner, K., and Wagner, H. -J.
MISSBRAUCH UND SUCHT IM HINBLICK AUF DEN VERKEHR. [Abuse and addiction in connection with traffic].
 In: Laubenthal, F., ed. *Sucht und Missbrauch: ein Kurzgefasstes Handbuch für Ärzte, Juristen, Pädagogen.* [Addiction and abuse: a short handbook for physicians, lawyers, teachers]. Stuttgart: Georg Thieme, pp. 402-423 (108 ref.), 1964.
 G – SEC – general – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – CNS – analg., antipyret. – anti-infectants – autocoids – indust. intox. – sed., hypnot. – tranquilizers – unclass. ther. agents – *CAAAL-0 A-1194.

Medico-legal aspects of drug use and abuse by drivers are presented. The symptoms and effects of analgesics, sedatives, hypnotics, stimulants, narcotics, tranquilizers, alcohol alone, and alcohol in combination with these drugs, are discussed in detail. It is pointed out that the high incidence of alcohol consumption and drug-taking in the population is creating a more and more common situation in which alcohol and drugs are ingested simultaneously. Very likely, synergism of alcohol and drugs will increasingly be the cause of traffic accidents. The significance of informing the health profession and the public about drug and traffic problems is stressed.

1409. Wahlström, G.
THE EFFECTS OF REPEATED LONG-TERM TREATMENTS WITH BARBITAL OR ALCOHOL ON A HEXOBARBITAL ANAESTHESIA THRESHOLD IN MALE RATS.
 Acta Physiol. Scand. (Stockholm), Suppl. 330: 71 (1 ref.), 1969.
 E – abst. – exp. comp. – cross-tol. – mammals – acute admin. – in vivo – CNS – sed., hypnot. – *CAAAL-0 B-0586.

In previous experiments, it was found that, after constant infusion of 0.25 mg hexobarbital/kg/sec into the tail vein of rats, and using as threshold the dose necessary to obtain a burst suppression of 1 sec or more in the EEG, treatment with 200 mg/kg/sec barbitol for 6 weeks caused an increase in the threshold dose, which disappeared within 1 week. In the present experiment, 2-6 week barbitol treatments, 3 months apart, were performed. In 1 test, 10% w/v alcohol in the drinking water for 16 weeks was substituted for the second barbitol treatment; the rats had access to alcohol for 2-1 hr periods/day. It was found that the second barbitol treatment increased hexobarbital thresholds 1 week after the end of treatment, and a new increase was seen 3 weeks after treatment. When alcohol was substituted for barbitol, a threshold increase 3 weeks after the end of treatment was also seen. It is concluded that the first barbitol treatment brought about a change which made the threshold increase after the second treatment more pronounced. A 3-month abstinence period did not alter the change.

1410. Wahlström, G.
INTERACTION OF ETHANOL AND HEXOBARBITAL IN UNTREATED AND LONG-TERM ETHANOL TREATED MALE RATS.
 Acta Pharmacol. (Copenhagen), 28, Suppl. 1: 90 (1 ref.), 1970.
 E – abst. – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vivo – CNS – barbiturates – *CAAAL-0 B-0999.

The effect of prior administration of 2 g/kg ethanol ip on the threshold (dose needed to obtain a burst suppression of 1 sec or more in the electroencephalogram) of 0.25 mg/kg/sec hexobarbital infusion iv in the rat was determined. The presence of ethanol reduced the hexobarbital threshold. A blood ethanol level (BEL) of 1.5-2.0 mg/ml on the rising part of the concentration curve reduced the hexobarbital threshold by about 50%, while corresponding blood levels on the falling part reduced threshold by only 25%. The difference in effects of similar ethanol concentrations indicated that an acute tolerance was induced. The time lag between brain and blood ethanol concentrations acts in the opposite direction. The 2 drugs were additive for 3-6 hr after ethanol, up to a BEL of 2.0-2.5 mg/ml on the falling part. In 2 other experiments, after chronic administration for 4 weeks of 10%

w/v ethanol, 1.0-2.0 mg/ml on the falling part of the blood ethanol curve had no influence on hexobarbital threshold. Threshold determination without ethanol was uninfluenced by chronic ethanol treatment.

1411. Wahlström, G.

CHANGES IN A HEXOBARBITAL ANESTHESIA THRESHOLD IN RATS INDUCED BY REPEATED LONG-TERM TREATMENT WITH BARBITAL OR ETHANOL.

Psychopharmacologia (Berlin), 19: 366-380 (21 ref.),

1971.

E – exp. cont. – DC (decrease) – mammals – chronic admin. – in vivo – CNS – barbiturates – sed., hypnot. – *CAAAL-0 B-1026.

The amount of iv hexobarbital needed to produce an EEG burst suppression of 1 sec or more was determined in male rats after chronic barbital and/or ethanol administration. Sleeping times were also recorded. At the end of administration of 200 mg barbital/kg/day ip for 5 weeks, the hexobarbital threshold was increased by 45%; thresholds were normal after about 1 week. At the end of a second barbital treatment, there was a similar, immediate, slightly more prolonged threshold increase. 3 weeks after the second treatment, there was a new increase in threshold. Sleeping times were unaffected. 10% w/v ethanol in drinking water, allowed twice/day for 16 weeks (average of 3-5 g ethanol/day), caused a gradual threshold increase which reached a max of 20% on days 9 and 10 after the end of treatment; 2 weeks after ethanol treatment, hexobarbital thresholds were normal. In an earlier group given 200 mg barbital/kg/day ip for 5 weeks, a second, slightly larger increase was seen about 3 weeks after the end of the ethanol treatment. The late changes in threshold after a second treatment appear to be due to a "summation" of changes induced by the 2 treatments. In this respect, ethanol and barbital are probably related, but, with respect to their effects on the threshold after interruption of chronic treatment, they are not identical.

1412. Wahlström, G., and Widerlöv, E.

INTERACTION AND ACUTE CROSS TOLERANCE BETWEEN ETHANOL AND HEXOBARBITONE IN THE RAT.

J. Pharm. Pharmacol. (London), 23(1): 58-60 (9 ref.),

1971.

E – exp. cont. – cross-tol. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – barbiturates – *CAAAL-15353 B-1027.

The correlation between the blood alcohol curve (BAC) and the threshold dose of hexobarbitone required to obtain an EEG burst suppression for 1 sec or more was investigated in male rats (350 g). The standard BAC resulting from 20% w/v ethanol ip, and the pre-ethanol hexobarbitone threshold, plus ensuing sleeping time, were determined, following which hexobarbitone was administered after ethanol (above doses). Threshold measurements were taken 1.25, 2.75, and 5.75 hr after ethanol injection (peak and descending part of the BAC). The same experiment was repeated on the same animals 2-3 weeks later, and threshold measurements were made 0.2, 0.25, and 0.4 hr after ethanol (ascending part of the BAC). There was a linear relationship between the BAC and the threshold dose on the descending part of the BAC. There was a slight deviation at the peak, while no linear relationship could be established for the ascending portion of the curve. Compared to the pre-ethanol measurement of 18.9 ± 1.3 min, post-ethanol sleeping times were approximately the same for the descending BAC, and were increased by 14.5 ± 2.7 and 18.0 ± 3.6 min for the peak and ascending BAC, respectively. It is concluded that cross tolerance does exist between ethanol and hexobarbitone.

1413. Wahren, H.

UNTERSUCHUNGEN ÜBER DIE EINWIRKUNG VON GEFÄSSTONIKA BEI AKUTER SCHLAFMITTEL- BZW. ALKOHOLVERGIFTUNG. [Investigations on the influence of cardiotonics in acute poisoning with soporifics or alcohol].

Z. Ges. Exp. Med. (Berlin), 99: 320-324 (4 ref.), 1936.
 G – exp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – absorp., distrib., stor. – CNS – G.I. tract – anesthetics – autonomic agents – sed., hypnot. – stimulants – *CAAAL-1181-B2 A-1195.

A cat, anesthetized with 1 cc of pernocton im plus 5 cc of ether, received 2 doses of 20 cc of 40% alcohol, 1/2 hr apart, by stomach tube. It was found that carbon dioxide (CO₂) absorption in the isolated small intestine was decreased by 50%. Iv and im injections of coramine, cardiazole, ephedrine, and sympathol had no effect on CO₂ absorption. Other studies are reported, but no ethanol was administered.

1414. Walker, G., and Boyd, P. R.

TETRAETHYL LEAD POISONING: REPORT OF A NON-FATAL CASE.

Lancet (London), 263: 467-469 (11 ref.), 1952.
 E – SEC – general – case hist. – DC (unspec.) – humans – mot. perform. – psychol. perform. – CNS – nerv. syst. – senses – miscellaneous – *CAAAL-0 A-1357.

3 cases of tetraethyl lead (TEL) poisoning (1 fatal) are described, 1 in detail. In 1 non-fatal case, a man who was foreman of a gang of men employed to remove TEL-containing sludge from storage tanks which had contained lead gasoline, failed to observe the stringent rule that a positive-pressure air-line respirator must be worn; consequently, the unprotected faces of both him and his men were occasionally splashed with sludge. After about 14 months, the man began to experience difficulty in concentrating and in falling asleep, and was increasingly worried about domestic matters. A heavy beer drinker, he abruptly stopped drinking. Within several weeks he was depressed and listless, and soon afterwards was admitted to hospital. The symptoms were: extreme worry, guilt, and a feeling of instability (although physical signs of anxiety were absent), a twitching of the nose, a feeling of something stuck in the throat, a creeping sensation in the region of the right ear and shoulder, transient blurring of vision, episodes of apparent *déjà vu*, insomnia, halitosis, metallic taste in the mouth, voracious appetite but loss of weight, mild sore throat, and occasional headaches. An extensive examination is described. The curious development of symptoms within a few days of stopping heavy beer drinking is noted by the authors, although no explanation is offered.

1415. Wallace, G. B.

ALCOHOL AS AN ANTIDOTE FOR CARBOLIC ACID.

New York University Bulletin of the Medical Sciences (Lancaster), 2(1): 58-63 (6 ref.), 1902.
 E – exp. cont. – DC (antidotal) – DC (unchanged) – humans – mammals – acute admin. – in vivo – absorp., distrib., stor. – CNS – skel., muscle, skin – anesthetics – *CAAAL-0 A-1196.

The author carried out experiments to determine the effect of alcohol applied after phenol on human skin and in animals. It was concluded that the application of alcohol must follow that of the carbolic acid within 5 min to completely remove the local effects of the acid. A rabbit and a cat were given a min lethal dose of carbolic acid, followed by 60% alcohol sc at intervals. Alcohol administration did not influence the course of the poisoning, and the animals died at approximately the same time as the controls. It is thus concluded that the antidotal action of alcohol is only a local one, and it is unable to check the effects of the absorbed acid.

1416. Wallgren, H.

BERUSNINGSTEST. [Intoxication test].

Alkoholpolitik (Helsinki), 19: 101-103 and 122-124 (2 ref.), 1956.
 S – ES – SEC – exp. cont. – mammals – in vivo – *CAAAL-8287-J2 A-1197.

This paper is concerned with the development of a method whereby the degree of intoxication from alcohol of a laboratory animal can be reliably measured. A tentative test is described and the criteria

are discussed. The proposed technique is considered suitable for determining whether or not drugs are synergistic with, or antagonistic to, alcohol.

1417. Wallgren, H.

PHYSIOLOGICAL ASPECTS OF ETHANOL ACTION IN STIMULATED BRAIN SLICES.
Acta Physiol. Scand. (Stockholm), 50(Suppl. 175): 151-152 (2 ref.), 1960 (2 ref.).
E – abst. – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – in vitro – acid-base, blood pH, elect. – nerv. syst. – *CAAAL-9487-B2 A-1198.

The mechanism of effect of alcohol on stimulated brain slices is discussed. "Like ethanol, some lipid-soluble analogues of acetylcholine block axonal conduction by depolarization. One compound tested was pyridine-aldoxamine-dodecyl iodide (PAD), the effects of which resemble those of ethanol. PAD and ethanol are synergistic. In terms of Nachmansohn's theory that the acetylcholine system provides an essential trigger mechanism for the excitation cycle of conducting membranes, the results may be interpreted as indicating that ethanol possibly interferes with this system in an unspecific manner."

1418. Wallgren, H.

COMPARISON OF THE EFFECT OF ETHANOL AND MALONATE ON THE RESPIRATION OF RAT BRAIN CORTEX SLICES.
Acta Physiol. Scand. (Stockholm), 49: 216-223 (13 ref.), 1960.
E – exp. cont. – DC (unchanged) – mammals – acute admin. – in vitro – dose resp. – respir. – *CAAAL-9487-B2 A-1448.

The effects of 2 inhibitors, ethanol and malonate, on respiration of electrically-stimulated and normal rat brain cortex slices were measured on Warburg manometers. The inhibitors were added at low concentrations, separately or together, to a phosphate-glucose saline sol. It was found that, within the concentration ranges employed (malonate— 10^{-4} to 8×10^{-4} M; ethanol— 8.7×10^{-2} to 1.96×10^{-1} M), the only effect on unstimulated tissue was a slight increase in respiration in the presence of ethanol. In electrically-stimulated tissue, both malonate and ethanol decreased respiration. The inhibiting action of ethanol appeared to be proportional to its concentration in the medium, and was greatly reduced by a fall in temperature, whereas malonate inhibition was only slightly reduced by a lower temperature. There was a marked difference in the concentrations of the 2 agents required to produce the same fall in oxygen uptake. When the inhibitors were present together, the effect of only 1 of them, that which had the greater action at the particular concentration, was obtained. This lack of synergism indicates that the mechanisms of inhibition by ethanol and malonate are independent.

1419. Wallgren, H.

EFFECTS OF ACETYLCHOLINE ANALOGUES AND ETHANOL ON THE RESPIRATION OF BRAIN CORTEX TISSUE *IN VITRO*.
Biochem. Pharmacol. (New York), 6: 195-204 (17 ref.), 1961.
E – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – in vitro – mot. perform. – acid-base, blood pH, elect. – blood comp., sites, lymph – nerv. syst. – anti-infectants – autonomic agents – musculoskel. agents – respir. agents – *CAAAL-9487-B2 A-1199.

In experiments with rat brain cortex slices, acetylcholine, curare, deca- and suxa-methonium did not affect the respiration. The acetylcholine analogues, pyridine-2-aldoximedodecyl iodide (PAD) and cetyltrimethylammonium bromide (CAB), caused a transient increase in the respiration of unstimulated tissue. Ethanol significantly increased the effect of atropine, PAD, CAB, and CPC (cetylpyridine chloride) on respiration in stimulated tissue. In intact rats, PAD and CAB potentiated the

action of ethanol on tilted plane performance. The results indicate that, as the synergism with ethanol is restricted to the stimulated tissue, ethanol may act directly on processes linked with the excitation cycle of conducting membranes.

1420. Wallgren, H., and Tirri, R.

STUDIES ON THE MECHANISM OF STRESS-INDUCED REDUCTION OF ALCOHOL INTOXICATION IN RATS.

Acta Pharmacol. (Copenhagen), 20: 27-38 (13 ref.),

1963.

E – exp. cont. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – blood lev. – mot. perform. – CNS – amphetamines – autonomic agents – *CAAAL-10665-J2

A-1200.

The effects of vigorous exercise and stress were tested in 5 experiments on rats. In 1 experiment, using a tilting-plane test for intoxication, 1 $\mu\text{g/g}$ adrenaline was given with and without 2 mg/g alcohol in 10% sol ip; 3 days later, the adrenaline-only rats received 5 μg amphetamine and 2 mg/g alcohol, and the alcohol-adrenaline rats received amphetamine only. Neither drug improved performance, but amphetamine-alcohol and adrenaline-alcohol both had a significant effect. Amphetamine had no influence on the blood alcohol level. In another test, 2 mg/g alcohol ip was administered to rats, followed 2 days later by a second dose of 3 mg/g ip; 3 days later, 1 group was given 5 μg amphetamine plus 3 mg/g alcohol ip. As compared to a previous swimming experiment with 2 mg/g alcohol alone, 3 mg/g alcohol had a smaller and shorter effect; amphetamine had no effect. The amphetamine-improved performance in the first experiment suggests that an arousal reaction is involved. The results are in agreement with the view that the brain-stem reticular activating system is sensitive to alcohol, but that, with moderate doses, the effect is largely reversible. Since the effect of exercise was diminished, and that of amphetamine blocked, after ip injection of 3 mg/g alcohol, it is concluded that there is a threshold dose of alcohol above which the arousal reaction becomes difficult to achieve.

1421. Wallgren, H.

OUABAIN-INDUCED DEPRESSION OF THE RESPIRATION OF ELECTRICALLY STIMULATED BRAIN SLICES IN PRESENCE AND ABSENCE OF ETHANOL.

Ann. Med. Exp. Biol. Fenn. (Helsinki), 41: 166-173 (21 ref.),

1963.

E – exp. cont. – DC (unchanged) – mammals – in vitro – dose resp. – acid-base, blood pH, elect. – CNS – cardiovasc. agents – *CAAAL-10719-B2

A-1201.

Ouabain at low concentrations depressed the respiratory response of rat brain cortex slices to electrical stimulation. Response to stimulation with 30 mM potassium chloride was partly inhibited by 5 μM ouabain. At 5 and 10 μM , ouabain slightly depressed respiration of unstimulated tissue. The effect of 1 and 2 μM ouabain in the presence of ethanol, at a concentration which by itself causes considerable depression of response to stimulation (0.9%), was not synergistic. Ethanol and ouabain seem to act on different respiration-limiting systems in the stimulated brain slices.

1422. Wallgren, H., Sammalisto, L., and Suomalainen, H.

PHYSIOLOGICAL EFFECT OF PHENETHYL ALCOHOL.

Institute of Brewing, Journal (London), 69: 418-420 (8 ref.),

1963.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – mot. perform. – CNS – metab. proc. – alcohols – *CAAAL-10721-D2

A-1449.

Of 9 groups of 12 mice each, 3 groups received 2.0 mg/g ethanol, 3 received the same ethanol dose plus 0.1, 0.2, or 0.4 mg/g phenethyl alcohol, and 3 received 1.3, 0.6, or 0.9 mg/g phenethyl alcohol only. The alcohols were administered by stomach tube as a 10% (v/v) sol. Performance was tested, using the tilting-plane technique as an index of sobriety, and measurements were made every 15 min up to 2 hr after alcohol ingestion. The molar intoxicating effect of phenethyl alcohol was found to

be 1.6-2.5 times stronger than ethanol, an effect slightly less than that of the propanols, and recovery from its effects was very rapid. No intoxication was seen with a dose of 0.1 mg/g phenethyl alcohol. The regression coefficient of performance impairment was significantly greater when phenethyl alcohol was administered together with ethanol, suggesting that the combined effect is simply additive. In separate experiments, toxicity and narcotic effects were studied. In contrast to ethanol, phenethyl alcohol had a sedative effect, and in large doses, a hypnotic effect. It is concluded that the amount of phenethyl alcohol found in beer (10-40 mg/l) is not hazardous to humans, if consumed in moderate amounts.

1423. Wallgren, H., and Barry, H.

DRUG ACTIONS IN RELATION TO ALCOHOL EFFECTS.

In: *Actions of Alcohol. II. Chapter 10.* Amsterdam: Elsevier,

pp. 621-714 (417 ref. in chapter) @ 1970.

E – review – congen. stud. – cross-tol. – DC (antidotal) – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – drug-dep. humans – mammals – other org. – absorp., distrib., stor. – acid-base, blood pH, elect. – blood comp., sites, lymph – cardiovasc. – CNS – G.I. tract – glands – liver, kidney – metab. proc. – nerv. syst. – respir. – senses – skel., muscle, skin – alcohols – amphetamines – analeptics – analg., antipyret. – anesthetics – anticonvulsants – antidepressants – anti-infectants – antispasmodics – autonomic agents – barbiturates – cardiovasc. agents – elect., water-bal. agents – hallucinogens – sed., hypnot. – stimulants – tranquilizers – unclass. ther. agents – *CAAAL-0 B-1000.

The experimental literature on alcohol-drug interaction is exhaustively reviewed. Reported experimental interactions, involving 270 separate references to published and unpublished studies, are tabulated according to: drug, subject species, criterion or measure, findings, and comment. In a section on the joint action of drugs and acutely-administered alcohol, the terminology and methodology, the reported modification of alcohol absorption and penetration, and the modification of alcohol metabolism or excretion, are discussed. In a second section, ethanol is compared with other drugs. A third section on chronic alcohol administration includes: changes in capacity to metabolize drugs, cross-tolerance to general anesthetics and barbiturates, and alcohol interaction with other agents. The fourth section, on drugs with special relationships to ethanol, deals with drugs producing intolerance to alcohol, and with ethanol interactions with other alcohols. The fifth section discusses the actions of components of alcoholic beverages, with respect to both acute and chronic effects. The chapter concludes with a comprehensive summary.

1424. Walsh, M. J., and Truitt, E. B., Jr.

CNS EFFECTS OF ACETALDEHYDE (CH₃CHO) AND ETHANOL (ETOH) AND INTERACTIONS WITH CATECHOLAMINES AND PSYCHOTROPIC DRUGS.

Pharmacologist (Washington), 11: 279 (O ref.),

1969.

E – abst. – exp. cont. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – CNS – metab. proc. – autonomic agents – enzymes – *CAAAL-0 B-0585.

The effects of acetaldehyde and ethanol on the CNS were studied in mice. Acetaldehyde was found to be a potent hypnotic which is more active than ethanol, and about 30 times more toxic. The hypnotic activity of acetaldehyde was dose-related with an ED₅₀ in mice of 275 mg/kg, giving a mean blood level of 108 µg/ml. However, the in vitro, non-enzymatic production of acetaldehyde by the brain constitutes a substantial error in reported values. The administration of 0.06 and 0.12 mM/kg of norepinephrine to mice prolonged ethanol sleeping-time by 200% and 300% of control values, respectively, without altering ethanol blood levels. Ethanol-induced hypnosis was unaffected by the 5 metabolites of norepinephrine, but was markedly potentiated by monoamine oxidase inhibitors. It is concluded that these findings fail to implicate the metabolites or the intermediate aldehyde of norepinephrine in ethanol effects, but may explain the enhanced depression recently observed between ethanol and certain antidepressants.

1425. Walter, U.

ÜBER DIE BEEINFLUSSBARKEIT DES ALKOHOLGEHALTES IM BLUT DURCH ARZNEIMITTEL. [The influence of medicaments on the blood alcohol level].

Deutsch. Z. Ges. Gerichtl. Med. (Berlin), 30: 243-256 (20 ref.), 1938.
G – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo
– blood lev. – absorp., distrib., stor. – metab. proc. – analg., antipyret. – *CAAAL-869-A1
A-1202.

9 human subjects were given alcohol (60 cc absolute alcohol in 30% sol in 10 min) only, or alcohol followed by po administration of 1 g aspirin, 1 g gardan, or 22 guttae neutragol. Blood alcohol levels were determined 1, 1 1/2, 2 1/2, 3, 4, 5, 6, and 8 hr after alcohol. It is concluded that neither aspirin nor neutragol influenced the blood alcohol level or the rate of oxidation; gardan may have had a slight effect on absorption, but not to any practical degree. Aspirin and gardan improved the subjective feeling, but neutragol was without effect.

1426. Walter, U.

UEBER DIE BEEINFLUSSBARKEIT DES ALKOHOLGEHALTES IM BLUT DURCH ARZNEIMITTEL. [The influence of medicaments on the blood alcohol level].

Dissertation, Medical Faculty of the University of Frankfurt, Germany, 14 pp. (23 ref.), 1938.
G – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo
– blood lev. – absorp., distrib., stor. – metab. proc. – analg., antipyret. – *CAAAL-0 A-1203.

9 human subjects were given alcohol (60 cc absolute alcohol in 30% sol in 10 min) only, or alcohol followed by po administration of 1 g aspirin, 1 g gardan, or 22 guttae neutragol. Blood alcohol levels were determined 1, 1 1/2, 2 1/2, 3, 4, 5, 6, and 8 hr after alcohol. It is concluded that neither aspirin nor neutragol influenced the blood alcohol level or the rate of oxidation; gardan may have had a slight effect on absorption, but not to any practical degree. Aspirin and gardan improved the subjective feeling, but neutragol was without effect.

1427. Wambsganss, E.

EXPERIMENTALPSYCHOLOGISCHE STUDIE ÜBER DIE

INTERFERENZWIRKUNGEN ZWEIER PSYCHOPHARMAKA. [Experimental psychological studies on the interference effects of two psychopharmacological drugs].

Arzneimittelforschung (Aulendorf), 15: 1063-1069 (11 ref.), 1965.
G – ES – exp. cont. – exp. comp. – DC (unchanged) – humans – acute admin. – in vivo – blood lev.
– mot. perform. – psychol. perform. – CNS – senses – autonomic agents – tranquilizers – *CAAAL-0
B-0480.

The interaction effects of a drug combination (haloperidol plus isopropamide—3 day medication), placebo, and alcohol (1 1/2 l 10.6% wine) were examined in a double-blind study on 18 human subjects by psychological performance tests. Performance was chiefly influenced by the effect of practice, and could be significantly disturbed neither by the alcoholic influence nor by the combined alcohol-drug effect, under the chosen test conditions. No potentiating or compensating influence of simultaneous alcohol administration on the effect of the drug on the organism was found.

1428. Wambsganss, E., and Bredenkamp, J.

EINE EXPERIMENTALPSYCHOLOGISCHE UNTERSUCHUNG ÜBER DIE WIRKUNG VON HALOPERIDOL IN NIEDRIGER DOSIERUNG BEI ALLEINIGER APPLIKATION UND IN VERBINDUNG MIT ALKOHOL. [An experimental psychological study on the effect of haloperidol in low dosage, given alone or in combination with alcohol].

Arzneimittelforschung (Aulendorf), 18(2): 238-243 (23 ref.), 1968.
G – ES – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans

– psychot. humans – acute admin. – chronic admin. – in vivo – blood lev. – psychol. perform. – CNS
– sed., hypnot. – tranquilizers – *CAAAL-13600-J1 B-1028.

A double-blind study was performed on healthy and neurotic humans, to determine the influence of haloperidol, alone and in combination with alcohol, on psychic and intellectual activity. 88 male and female subjects, aged 20-26 yr, were divided into 8 groups, of which 4 received haloperidol (2 x .50 mg po) and 4 received a placebo, for a 14-day period. On the day of administration of the last tablet, 2 of the haloperidol and 2 of the placebo groups consumed 1/2 l of wine within 15 min; 15 min after wine ingestion, psychological tests and a self-rating test were performed. The other groups performed the same tests without alcohol. It was found that the low haloperidol dosage had a lightly sedative action, and produced no noticeable side effects in normal subjects, whether administered alone or with alcohol. After receiving alcohol, however, the neurotic subjects showed a diminished efficiency on the "semantic classification" test, as well as a general depression of mood. It is concluded that haloperidol is well tolerated in humans, and that the side effects of its combination with alcohol on the mood of neurotic patients is due to a synergistic interaction.

1429. Wandrey, D., and Leutner, V.

VERKEHRSMEDIZIN. I. PSYCHOPHARMACA UND VERKEHRSSICHERHEIT. II. PSYCHOPHARMACA UND ALKOHOL. [Traffic medicine. I. Psychopharmacological drugs and traffic safety. II. Psychopharmacological drugs and alcohol].

In: Wandrey, D., et al., eds. *Neuro-Psychopharmaca in Klinik und Praxis*. [Neuro-psychopharmacological drugs in clinic and practice]. Stuttgart: Shattauer, pp. 135-143 (91 ref.), 1965.

G – review – DC (add., infra-add., unspec. incr.) – med.-leg. – mot. vehic. – humans – blood lev. – CNS – autocoids – barbiturates – hallucinogens – sed., hypnot. – stimulants – tranquilizers – *CAAAL-0 B-0481.

In this chapter, the potentiating effects of tranquilizers and other psychotropic drugs on alcohol, barbiturates, and other centrally acting substances are briefly discussed, especially in relation to traffic medicine. The possibility of enhanced or altered alcohol effects has to be taken into consideration when examining traffic offenders in whom a drug influence might still be present.

1430. Wangel, J.

ALCOHOL, ROAD TRAFFIC, AND DRUGS IN DENMARK, 1960.

In: Havard, J.D.J., ed. *Alcohol and Road Traffic*. Proceedings of the Third International Conference on Alcohol and Road Traffic at London, September 3-7, 1962. London: British Medical Ass., pp. 162-165 (1 ref.), 1963.

E – stat. surv. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – med.-leg. – mot. vehic. – humans – blood lev. – CNS – analg., antipyret. – barbiturates – tranquilizers – *CAAAL-0 A-0029.

A study of 6,067 medico-legal examinations in Denmark showed that the average blood alcohol concentration was almost the same for drivers using drugs as for those not using drugs. Drug use was more prevalent in the group which had alcohol in the blood than in the non-drinking drivers; this was especially true for analgesic, antipyretic, and antirheumatic drugs, for hypnotics (especially barbiturates), and for psychopharmacological drugs. The comparison of a group of 18 chronic meprobamate-users, ages 31-45 (average blood alcohol level 1.46°/oo, with 109 non-drug users, ages 31-45 (average blood alcohol level 1.49°/oo, showed that meprobamate consumption had not potentiated the effect of alcohol, as far as driving ability was concerned.

1431. Warburg, O.

ÜBER HEMMUNG DER BLAUSÄUREWIRKUNG IN LEBENDEN ZELLEN. [Inhibition of hydrocyanic acid action in living cells].

Hoppe Seyler Z. Physiol. Chem. (Berlin), 76: 331-346 (14 ref.), 1911.
 G – exp. – DC (add., infra-add., unspec. incr.) – other org. – in vitro – blood comp., sites, lymph
 – metab. proc. – neoplast. agents – *CAAAL-0 A-1204.

Oxidation inhibitors, such as different alcohols, formaldehyde, urethane, etc., are additive in their inhibiting action on the oxidation process, but the simultaneous effect of hydrocyanic acid (HCN) and alcohol is less than the sum total of both effects. If the degree of concentration is carefully calculated, the effect of both compounds will be found to be even less than the effect of HCN alone. Using young erythrocytes of geese, it could be demonstrated that the oxidation process in cells treated with potassium cyanide (KCN), 0.1 mM/l sol, could be increased 20-50% by adding 70 mM/l ethanol sol, 45 mM/l butyl alcohol sol, or 18 mM/l amyl alcohol sol. The experiments are described in detail, and graphs and tables are presented.

1432. Waris, E.

EFFECT OF ETHYL ALCOHOL ON SOME COAGULATION FACTORS IN MAN DURING ANTICOAGULANT THERAPY.

Ann. Med. Exp. Biol. Fenn. (Helsinki), 41: 45-53 (10 ref.), 1963.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – blood lev. – blood comp., sites, lymph – liver, kidney – coagulants – *CAAAL-10717-B1 A-1205.

The effects of alcohol on coagulation factors in normal persons and patients with coronary disease were investigated. In healthy subjects given alcohol po, the alcohol had no definite effect on the thromboplastin time. To patients with coronary disease, alcohol was administered by drip infusion or po during anticoagulant therapy, and the blood coagulation activity was studied before and after the alcohol infusion. A slight falling tendency in the thrombotest values was observed in some cases. A certain caution in the use of alcohol by patients under anticoagulant therapy is advisable, since individual differences in its effect were observed, but, in general, incidental drinking does not seem to constitute a risk for such persons.

1433. Wartburg, J. -P. von, and Röthlisberger, M.

DER EINFLUSS VON FUSELÖL AUF DEN OXYDATIVEN ABBAU DES AETHYLALKOHOLS. [The influence of fusel oil on the oxidative metabolism of ethyl alcohol].

In: *Twenty-sixth International Congress on Alcohol and Alcoholism, Abstracts*. Stockholm, Sweden, August 1-5, pp. 518-519 (0 ref.), 1960.

G – abst. – exp. comp. – presentation – DC (add., infra-add., unspec. incr.) – mammals – in vitro – liver, kidney – metab. proc. – alcohols – *CAAAL-0 A-1206.

The influence of n-propanol, iso-butanol, amyl alcohol, and fusel oil on the oxidation of 1-C¹⁴-ethanol was investigated in rat liver sections and in pure alcohol dehydrogenase. It was established that low concentrations of fusel oil and its principal individual components (a small percentage of the ethanol concentration) inhibit the oxidation of 1-C¹⁴-ethanol to carbon dioxide in rat liver slices. A corresponding inhibition of 1-C¹⁴-ethanol metabolism was observed with n-propanol. It is assumed that the toxic effect of fusel oil is potentiated in interaction with ethanol. The fusel oil content of some spirits prolongs the oxidation time of ethanol, and consequently increases its toxic effect. In view of this, stringent control is advocated to safeguard against the presence of higher alcohols and fusel oils in spirit beverages.

1434. Wasik, A., and Bryś, J.

PSYCHOPATOLOGICZNE ZESPOŁY KOMBINOWANEGO ZATRUCIA HYDRAZYDEM KWASU IZONIKOTYNOWEGO I ALKOHOLEM ETYLOWYM. [Psychopathological syndromes of a combined isonicotinic acid hydrazide-ethyl alcohol intoxication].

Psychiat. Pol. (Gdansk), 1(2): 165-169 (18 ref.), 1967.

Po – ES – RS – general – DC (add., infra-add., unspec. incr.) – DC (sensit.) – humans – psychol. perform. – CNS – metab. proc. – anti-infectants – *CAAAL-0 B-0482.

3 cases of reactions after combined intake of isoniazid and alcohol are reported. In 1 case, a psychosis of a type of protracted exogenous syndrome followed a single ingestion of 2,000 mg isoniazid and 200.0 mg of brandy. A 10-day schizophrenia-like exogenous reaction was seen in a patient after ingestion of 2,000 mg isoniazid and several glasses of wine, and intolerance to alcohol, in the form of vegetative disturbances similar to the disulfiram reaction, and in which intoxication was greatly out of proportion to the amount of alcohol ingested, occurred in a patient on isoniazid therapy who took 50.0 mg brandy. The author remarks that isoniazid is not only an inhibitor of monoamine oxidase, but also acts on other enzymes that participate in the oxidation of alcohol in the organism, whereby the toxic action of ethanol on the CNS is enhanced and there is a lowered tolerance to alcohol. Symptoms of deep intoxication then appear more rapidly, and psychopathic disturbances may occur.

1435. Wasik, A.

WPŁYW NIEKTÓRYCH LEKÓW NA PRZEBIEG UPICIA ALKOHOLEWEGO. [The influence of some drugs on the course of alcohol intoxication].

Problemy Alkoholizmu (Warsaw), 3/16(3): 6-7 (14 ref.),

1968.

Po – ES – general – DC (add., infra-add., unspec. incr.) – DC (sensit.) – CNS – metab. proc. – analg., antipyret. – enzymes – *CAAAL-0 B-0483.

The author discusses various classes of drugs, from the viewpoint of their influence on the course of alcohol intoxication. The interactions include: the ataractic and neuroplegic drugs, which potentiate the effects of alcohol; the MAO-inhibitors, which can induce psycho-motor excitation when taken during insobriety; and drugs like amidopyrine, butazolidine, and the anti-diabetic sulphonamides, that interfere with the metabolism of alcohol, thus raising the level of acetaldehyde, and inducing an antabuse-alcohol type of reaction.

1436. Watkins, W. D., Goodman, J. I., and Tephly, T. R.

EFFECT OF PYRAZOLE ON METHANOL AND ETHANOL OXIDATION.

Fed. Proc. (Bethesda), 28(2): 546 (0 ref.),

1969.

E – abst. – exp. – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – in vitro – liver, kidney – metab. proc. – *CAAAL-0 B-0484.

Pyrazole, a potent inhibitor of liver alcohol dehydrogenase (ADH), was tested for its ability to affect the oxidation of methanol and ethanol in vitro and in vivo. ADH from rat, monkey, and human liver was inhibited by pyrazole in vitro. Moreover, an 85% inhibition of ethanol-1-¹⁴C (91 g/kg) oxidation to ¹⁴CO₂ was observed in vivo in the rat. Methanol-¹⁴C (1 g/kg) oxidation was inhibited by about 50% over 4 hr after the administration of pyrazole (200 mg/kg) in the rat. In the monkey, methanol-¹⁴C oxidation of ¹⁴CO₂ was greatly impaired, a finding which is in agreement with previous studies showing that ADH was the major catalyst for methanol oxidation in the monkey. These observations suggest that pyrazole may be useful in the treatment of methanol poisoning in man.

1437. Watkins, W. D., Goodman, J. I., and Tephly, T. R.

INHIBITION OF METHANOL AND ETHANOL OXIDATION BY PYRAZOLE IN THE RAT AND MONKEY *IN VIVO*.

Molec. Pharmacol. (New York), 6(5): 567-572 (19 ref.),

1970.

E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – species or sex diff. – liver, kidney – alcohols – *CAAAL-15236 B-1029.

Studied were the effects: of pyrazole on crystalline catalase activity, of pyrazole and aminotriazole on rat hepatic catalase activity in vivo, of pyrazole on peroxidative methanol oxidation in vitro, of

pyrazole (2.94 mmoles/kg ip, 15 min before ethanol) on ethanol-1-¹⁴C (2.1 mmoles/kg ip) oxidation in vivo in rats, of pyrazole on methanol-¹⁴C oxidation in vivo in rats and monkeys, of ethanol (5.43 mmoles/kg ip, injected 8 hr after methanol) and pyrazole (0.735 mmoles/kg ip, injected 4 hr after methanol) on methanol-1-¹⁴C (32.3 mmoles/kg ip) oxidation in vivo in the monkey, and of ethanol (5.43 mmoles/kg ip, injected 4 hr after methanol) on methanol-¹⁴C (32.3 mmoles/kg ip) oxidation in the monkey. Pyrazole had no effect on the catalase-peroxidative system, but exerted a pronounced inhibition on liver alcohol dehydrogenase and ethanol oxidation (80%) in the rat. In rats given methanol, pyrazole exerted a lesser effect, but an 80% inhibition of in vivo methanol oxidation was seen in the monkey. Low doses of ethanol plus low pyrazole doses depressed methanol oxidation to about 20%, and ethanol alone inhibited methanol oxidation by about 50%. The results confirm that the catalase-peroxidative system plays a major role in methanol metabolism in rats, while, in the monkey, alcohol dehydrogenase is the major catalyst for methanol oxidation.

1438. Watts, J., Cassidy, P., Byron, W., Reilly, J., and Krop, S.
 ETHANOL AUGMENTATION OF DESMETHYLIMIPRAMINE EFFECTS IN THE RAT.
 Toxic. Appl. Pharmacol. (New York), 11(2): 372-377 (20 ref.), 1967.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. – in vivo – CNS
 – G.I. tract – metab. proc. – antidepressants – *CAAAL-0 B-0485.

Osborne-Mendel rats of both sexes (250-300 g) received an aqueous sol of 10 mg/ml of desmethylimipramine (DMI), at doses of 15, 25, and 50 mg/kg, followed 3 hr later by 3 g/kg 50% ethanol; all sol were given daily by stomach tube, 7 days/week. Ethanol augmentation of DMI toxicity by alcohol at the 50 and 25 mg/kg dose levels was shown by changes in drug intake, survival time, and number of survivors. At the 15 mg/kg dose level, the animals receiving DMI alone were similar to the animals receiving DMI plus alcohol in drug intake, survival time, and number of survivors. The incidence of bloating for the combination group was significantly greater than for the controls.

1439. Wax, J., Ellis, F. W., and Lehman, A. J.
 ABSORPTION AND DISTRIBUTION OF ISOPROPYL ALCOHOL.
 Fed. Proc. (Bethesda), 8: 344 (0 ref.), 1949.
 E – abst. – exp. – DC (decrease) – mammals – acute admin. – in vivo – absorp., distrib., stor. – G.I.
 tract – alcohols – *CAAAL-0 A-1207.

The gastrointestinal absorption by isopropyl alcohol, as affected by site and extent of absorbing surface, concentration, time, successive doses, and systemic ethyl alcohol was studied in dogs. Increased absorption area resulted in increased tissue concentration, but did not influence total absorption. Ethyl alcohol, administered iv, markedly decreased the intestinal absorption of isopropyl alcohol.

1440. Wayne, E. J.
 PHARMACOLOGICAL ASPECTS OF BARBITURATE INTOXICATION.
 J. Forensic Med. (Johannesburg), 1(3): 172-174 (5 ref.), 1954.
 E – SEC – general – DC (add., infra-add., unspec. incr.) – humans – blood lev. – CNS – liver, kidney
 – respir. – barbiturates – *CAAAL-7111-Z27 A-1208.

Barbiturate intoxication is discussed from the standpoint of alcohol-barbiturate interaction, dose variation, symptomatology, and treatment of acute poisoning. It has been shown that alcohol potentiates the action of barbiturates, and that both the lethal dose and the anesthetic dose of a barbiturate are less if alcohol is present in the blood and tissues. Morphine also potentiates the action of barbiturates on the respiratory center.

1441. Wayne, E. J.

ALCOHOL AND DRIVING—THE PHARMACOLOGICAL BACKGROUND.

In: Havard, J.D.J., ed. *Alcohol and Road Traffic*. Proceedings of the Third International Conference on Alcohol and Road Traffic at London, September 3-7, 1962. London: British Medical Ass., pp. 113-118 (21 ref.), 1963.

E – SEC – general – presentation – DC (supra-add. incr.) – DC (add., infra-add. unspec. incr.) – humans – CNS – mot. perform. – psychol. perform. – autocoids – barbiturates – tranquilizers – *CAAAL-0 A-1209.

The absorption, distribution, and elimination of alcohol; urine and breath analysis; the action of alcohol on the nervous system; synergism and potentiation of alcohol by drugs; and alcohol tolerance are discussed. The distinction between synergism and potentiation is pointed out. The evidence for alcohol-barbiturate synergism or potentiation is conflicting; recent research by several authors is reviewed. There is evidence that additive effects, or even potentiation, may occur between alcohol and non-barbiturate hypnotics, tranquilizers, and antihistamines.

1442. Weatherby, J. H., and Clements, E. L.

CONCERNING THE SYNERGISM BETWEEN PARALDEHYDE AND ETHYL ALCOHOL.

Quart. J. Stud. Alcohol (New Haven), 21(3): 394-399 (21 ref.), 1960.

E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – sed., hypnot. – *CAAAL-9433-D2 A-1210.

The LD₅₀'s of alcohol and paraldehyde were determined in albino male mice (20-30 g), and found to be 7.00 ± 0.047 g/kg absolute ethanol and 1.77 ± 0.024 g/kg paraldehyde. All sol were adjusted so that 1 cc was required for 30 g body wt, and all injections were ip. Various fractions of the LD₅₀ of each substance were given alone and in combination with corresponding fractions of the other substance. It was found that no dose of alcohol or paraldehyde from 25 to 75% of the LD₅₀ produced death, but, in combination, the mortality ranged from 8 to 89%, depending on the dosages. The degree of additive action of all doses was less than 100%. With ip injections, a progressive decrease in additive action with an increase in the time interval between administration of paraldehyde and alcohol was noted.

1443. Webb, W. R., and Degerli, I. U.

ETHYL ALCOHOL AND THE CARDIOVASCULAR SYSTEM.

J.A.M.A. (Chicago), 191: 1055-1058 (17 ref.), 1965.

E – SEC – exp. comp. – congen. stud. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – cardiovasc. – barbiturates – *CAAAL-10856-D2 B-1001.

The effects of alcohol on cardiovascular hemodynamics and coronary flow were investigated in 3 groups of healthy mongrel dogs which had been anesthetized with minimal doses of thiopental sodium, supplemented by succinylcholine chloride. Group 1 (20 dogs) received 0.5 g/kg 70% ethanol (25% by vol in saline) iv within 5-10 min, group 2 (16 dogs) was given 1.5 g/kg ethanol iv within 15-25 min, and group 3 (5 dogs) received 5 g/kg ethanol iv within 30-45 min. 8 dogs in group 1 received bonded 7 yr-old bourbon as alcohol. Alterations in cardiac output, systemic pressure, stroke work, pulse, coronary flow, and coronary and peripheral resistance were measured. The decreased myocardial efficiency induced by alcohol in dogs under barbiturate anesthesia, noted by other authors, was confirmed. Cardiac output and stroke work increased markedly, paralleling the dose of alcohol. Systemic pressure was increased by 25% in group 2, while pulse remained unchanged in all groups. Coronary flow was significantly diminished in groups 1 and 2. Coronary resistance increased in all groups, while peripheral resistance decreased in group 2 and was unchanged in group 1. No difference was noted in the effects produced by the alcohol and the bourbon.

1444. Webb, W. R., Degerli, I. U., Cook, W. A., and Unal, M. O.
ALCOHOL, DIGITALIS AND CORTISOL, AND MYOCARDIAL CAPACITY IN DOGS.
 Ann. Surg. (Philadelphia), 163(6): 811-817 (22 ref.), 1966.
 E – exp. cont. – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo
 – dose resp. – blood lev. – cardiovasc. – barbiturates – cardiovasc. agents – musculoskel. agents –
 *CAAAL-0 B-0486.

The effects of alcohol on the myocardial functional capacity of the dog were investigated. Group 1 received alcohol (0.5, 1.5, or 5 g/kg iv) only. Group 2 received 7 γ /kg ouabain iv, 1 hr prior to or during administration of 1.5 or 5 g/kg alcohol, or 50 mg hydrocortisone prior to 1.5 g/kg alcohol. Group 3 received 1.5 g/kg ethanol iv, followed by 7 γ /kg ouabain iv or 50 mg hydrocortisone iv. Myocardial function curves were determined. In group 1, myocardial function curves showed some deterioration in every animal, even with the 0.5 g/kg dose, with marked gross heart dilatation. In group 2, ouabain or cortisol protected the heart when given either prophylactically or simultaneously with alcohol; however, the drugs were unable to protect the myocardium from damage. In group 3, therapeutic application of ouabain and cortisol restored the deteriorated heart function curves, and the dilatated condition was corrected; again, neither drug was effective against the 5 g/kg alcohol dose.

1445. Weber, J.
EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE PROPHYLAKTISCHE UND THERAPEUTISCHE WIRKSAMKEIT DER GOTHANIA-ANTIALKOHOL-PASTILLEN.
 [Experimental investigations on the prophylactic and therapeutic efficiency of gothania antialcohol tablets].
 Dissertation, Medical Faculty of the University of Heidelberg, Germany, 19 pp. (18 ref.), 1940.
 G – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – CNS – hormones,
 hormone antag. – stimulants – *CAAAL-0 A-1211.

1 healthy volunteer received, on 4 different days, 4 bottles of beer totalling 2.8 l, with an alcohol content of 41 g/l. On 4 other days, he received the same amount of alcohol and 6 “gothania” sobering tablets (chemical composition unknown). The blood alcohol levels were determined by the Widmark method. The gothania tablets had no influence on the blood alcohol levels or on the alcohol-impaired performance. It is concluded that the commercial preparation has neither a prophylactic nor a therapeutic effect on the action of alcohol, and it increases the unpleasant after-effects.

1446. Wei, E., Wong, L. C. K., and Hine, C. H.
SELECTIVE POTENTIATION OF CARBON TETRACHLORIDE HEPATOTOXICITY BY ETHANOL.
 Arch. Int. Pharmacodyn. (Gand), 189(1): 5-11 (22 ref.), 1971.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – CNS – liver, kidney – metab. proc. – anesthetics – barbiturates – *CAAAL-0
 B-1030.

Rats (110-150 g) received glucose + corn oil, ethanol (50% v/v po) + corn oil, glucose + carbon tetrachloride (CT—0.1 ml/100 g in corn oil sc), or ethanol (0.5 ml/kg sc) + CT (above dose, given 18 hr after ethanol); mice (20-30 g) received glucose + corn oil, ethanol (50% v/v po) + corn oil, glucose + chloroform (0.4 ml/100 g in corn oil sc), ethanol (5 g/kg po) + chloroform (above dose, given 18 hr after ethanol), ethanol (above dose) + corn oil, or ethanol (above dose) + CT (0.4 ml/100 g in corn oil sc, given 18 hr after ethanol). Drug biotransforming capacity was assessed by measuring sleeping time after administration of sodium hexobarbital (100 mg/kg ip at 20 mg/ml concentration, given 2 hr after CT) to rats, or sodium pentobarbital (60 mg/kg ip at a 30 mg/ml concentration, given 2 hr after CT or 24 hr after chloroform) to mice. CT and chloroform significantly prolonged barbiturate sleeping time; ethanol did not affect barbiturate sleeping time, nor potentiate the effect

thereon of CT and chloroform. Hexobarbital plasma levels at the time of awakening were not altered by ethanol, CT, or ethanol + CT. Ethanol produced a dose-dependent potentiation of serum glutamic-pyruvate transaminase (SGPT) response to CT in rats. Liver triglyceride was significantly elevated in rats given CT or ethanol + CT; ethanol pretreatment did not alter the liver triglyceride response to CT. It is concluded that ethanol selectively potentiates CT hepatotoxicity, affecting SGPT activity, but not triglyceride accumulation or depressed barbiturate metabolism.

1447. Wei, E., Wong, L. C. K., and Hine, C. H.
POTENTIATION OF CARBON TETRACHLORIDE HEPATOTOXICITY BY ETHANOL AND COLD.
 Toxic. Appl. Pharmacol. (New York), 18(2): 329-334 (24 ref.), 1971.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – glands – liver, kidney – metab. proc. – unclass. ther. agents – *CAAAL-0 B-1031.

Carbon tetrachloride (CT) hepatotoxicity was investigated in ethanol-pretreated rats and in rats exposed to cold (18 hr at 4°C). 50% v/v ethanol (4 g/kg) was given by stomach tube; controls received isocaloric glucose. CT (0.25 ml/kg, 1:4 v/v in corn oil) was given sc 18 hr after ethanol, cold exposure, or warmth (31-33°C), and CT controls received an equal vol of corn oil. The animals were decapitated 24 hr after CT, and serum and liver samples were obtained for analysis. Serum glutamic-pyruvate transaminase (SGPT) and liver triglyceride levels were used as indices of hepatotoxicity. In male and female rats, the SGPT response to CT was increased after ethanol or cold exposure. Liver triglyceride accumulation after CT, however, was not enhanced by either condition. Potentiation of the SGPT activity by ethanol was blocked by pentolinium bitartrate (a ganglionic blocking agent) and by warmth. Thyroidectomy did not block potentiation by ethanol. The role of the pituitary gland in the potentiation was not clarified because of increased mortality in hypophysectomized rats given ethanol. It is suggested that an ethanol-induced norepinephrine release results in increased liver susceptibility to CT.

1448. Weinig, E., and Schwerd, W.
ALKOHOL-BARBITURAT-SYNERGISMUS. [Alcohol-barbiturate synergism].
 Fortschritte der Medizin (Berlin), 74(19): 497-499 (5 ref.), 1956.
 G – review – DC (add., infra-add., unspec. incr.) – humans – mammals – CNS – analg., antipyret. – barbiturates – sed., hypnot. – stimulants – *CAAAL-8820-D3 A-1212.

The literature on alcohol-barbiturate synergism is reviewed. Animal experiments and observations on man are discussed. The authors of the review agree that there is a synergism between alcohol and the barbiturates, but question the opinion that it is a potentiating synergism. They stress that barbiturate narcosis should be used with the utmost caution in traffic accidents in which the possibility of previous alcohol consumption cannot be ruled out.

1449. Weiss, B., and Laties, V. G.
EFFECTS OF AMPHETAMINE, CHLORPROMAZINE, PENTOBARBITAL, AND ETHANOL ON OPERANT RESPONSE DURATION.
 J. Pharmacol. Exp. Ther. (Baltimore), 144(1): 17-23 (23 ref.), 1964.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – psychol. perform. – CNS – amphetamines – elect., water-bal. agents – *CAAAL-11098-J2 A-1213.

Male Bazenji dogs were trained to press a button with their noses, receiving a food reward after 1 min nose-pressing. At intervals of several days, amphetamine sulphate, chlorpromazine, pentobarbital, ethanol, amphetamine plus pentobarbital, and amphetamine (0.5 mg/kg sc) plus ethanol (2 g/kg by stomach tube) were administered, and the frequency and duration of dose response were deter-

mined. Amphetamine produced a progressive increase in the number of response/reward and the number of short (less than 1 sec) responses, as the dose was increased. The dose-response function for ethanol was less steep than for amphetamine, but, at the highest dose (2.0 g/kg), there was a clear increase in the number of responses/reward and in the number of short responses. Amphetamine plus ethanol produced a striking increase in dose-response, and the usually docile animals, despite signs of ataxia, became extremely hypertensive, and howled, whined, and struggled against the leash.

1450. Weiss, B., and Coen, G.

EFFECT OF ETHANOL ON ETHYLENE GLYCOL OXIDATION BY MAMMALIAN LIVER ENZYMES.

Enzym. Biol. Clin. (Basel), 6(4): 297-304 (15 ref.),

1966.

E – exp. – DC (decrease) – mammals – in vitro – liver, kidney – metab. proc. – indust. intox. – *CAAAL-0 B-0487.

The in vitro inhibition of ethylene glycol oxidation by ethanol was investigated. It was found that relatively small concentrations of ethanol inhibited ethylene glycol oxidation by horse liver alcohol dehydrogenase, beef liver catalase, and crude rat liver homogenates. The inhibition of alcohol dehydrogenase was of the competitive type. The major effect of ethanol on the rat liver homogenates was to decrease glycolaldehyde formation. The results provide a theoretical, biochemical basis for the use of ethanol in ethylene glycol poisoning, since by blocking the oxidation and formation of highly toxic metabolites of the latter, the toxicity of ethylene glycol in mammals may thus be lowered.

1451. Weiss, G. B.

THE ACTIONS OF CALCIUM, COCAINE, AND ETHANOL UPON POTASSIUM EFFLUX AND MECHANICAL RESPONSES IN SMOOTH MUSCLE.

Ph.D. Thesis, Graduate School of Vanderbilt University, Nashville, Tennessee, U.S.A., 126 pp. (153 ref.),

1962.

E – exp. cont. – exp. comp. – DC (decrease) – mammals – in vitro – acid-base, blood pH, elect. – G.I. tract – skel., muscle, skin – autonomic agents – elect., water-bal. agents – *CAAAL-0

A-1214.

The actions of calcium ion were investigated in resting and drug-excited smooth muscle. On the assumption that cocaine and ethanol may act by blockade of calcium ion, the actions of these 2 local anesthetics were contrasted with those of calcium. Both pilocarpine and acetylcholine were used as stimulatory drugs. The isolated longitudinal smooth muscle of the guinea pig ileum was employed in all experiments. The parameters measured were isotonic contractions and potassium (K^{42}) fluxes—the latter serving as an index of membrane permeability. Acetylcholine- and pilocarpine-induced smooth muscle contractions were prevented by ethanol in normal Tyrode's sol. The inhibition of an acetylcholine-induced contraction by ethanol could be largely reversed by addition of calcium, although, at the concentrations of calcium and ethanol used, the inhibition of a pilocarpine-induced contraction was not reversed.

1452. Weiss, L. R., and Orzel, R. A.

ENHANCEMENT OF TOXICITY OF ANTICHOLINESTERASES BY CENTRAL DEPRESSANT DRUGS IN RATS.

Toxic. Appl. Pharmacol. (New York), 10(2): 334-339 (13 ref.),

1967.

E – exp. cont. – exp. comp. – DC (decrease) – DC (add., infra-add., unsp. incr.) – DC (unchanged) – mammals – acute admin. – in vivo – dose resp. – CNS – respir. – *CAAAL-0 B-0488.

To investigate the possibility that central-depressant drugs enhance the acute toxicity of anticholinesterases in rats, the pesticides, carbaryl and parathion, were administered parenterally to animals pretreated with chlorpromazine, meprobamate, reserpine, chloriazepoxide, hexobarbital,

phenobarbital, and alcohol. Comparisons were made between carbaryl and carbaryl-drug combinations, and between parathion and parathion-drug combinations, using mortality as an index of toxicity. Ethanol (1500 mg/kg as a 25% sol) was administered ip to 60 female rats, 30 min before injections of 25, 50, or 100 mg of carbaryl/kg, or of 2, 4, or 8 mg of parathion/kg. Ethanol had no significant effect on the toxicity of carbaryl at any dose; it slightly increased the toxic effects of parathion at the 4 mg dose, and, at the 8 mg dose, it slightly decreased the mortality rate.

1453. Weissenberger, R.

EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE BEEINFLUSSUNG VON BLUTALKOHOLGEHALT UND TRUNKENHEIT DURCH „PEKASIN“. [Experimental investigations of the influence of Pekasin on the blood alcohol level and on intoxication].

Dissertation, Medical Faculty of the University of Heidelberg, Germany, 19 pp. (4 ref.), 1940.
G – exp. cont. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – mot. perform. – cardiovasc. – CNS – amphetamines – analg., antipyret. – anti-infectants – autonomic agents – barbiturates – hormones, hormone antag. – stimulants – *CAAAL-0 A-1215.

The sobering-up drug, “pekasin”, contains extract of cola, lecithin, magnesia usta, magnesium carbonate, animal charcoal, and other substances. Human subjects drank port wine to achieve a blood alcohol level of 0.56 to 1.1°/oo. To test performance, the subjects had to put rings on a bar, under the influence of alcohol, alone and in combination with pekasin (3 tablets). Pekasin had no effects on the blood alcohol level, as compared to the control value. Moreover, no improvement could be observed in objective or subjective performance of the test subjects, or in diminution of intoxication symptoms. Pekasin accelerated heart action, and caused disturbed sleep in the subjects.

1454. Wells, H. S.

A QUANTITATIVE STUDY OF THE ABSORPTION AND EXCRETION OF THE ANTHELMINTIC DOSE OF CARBON TETRACHLORIDE.

J. Pharmacol. Exp. Ther. (Baltimore), 25: 235-273 (24 ref.), 1925.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – dose resp. – absorp., distrib., stor. – G.I. tract – anti-infectants – tranquilizers – *CAAAL-0 A-1216.

A quantitative study of the absorption of the therapeutic anthelmintic dose of carbon tetrachloride (CCl₄) from the intestinal tract of the dog, and of the CCl₄ excretion in the expired air of dogs and humans was made, and an explanation of the effects of alcohol and magnesium sulphate was sought. The absorption of 3 cc CCl₄ from isolated intestinal loops of adult dogs was studied when the drug was injected into the gut together with 20 or 100 cc 50% alcohol or with 50 cc 97% alcohol. It was found that the 50% alcohol caused no constant increase in the absorption rate. On the other hand, the 97% alcohol caused a marked acceleration during the first 2-4 hr. An explanation of this phenomenon is offered.

1455. Werkgartner, A.

VERFÄLSCHT EVIPAN DEN BLUTALKOHOLWERT? [Does evipan falsify the blood alcohol value?].

Beitr. Gerichtl. Med. (Vienna), 20: 73 (0 ref.), 1955.
G – exp. cont. – DC (unchanged) – med.-leg. – humans – acute admin. – in vivo – blood lev. – CNS – barbiturates – *CAAAL-7410-U3 A-1217.

A Widmark value of 1.7°/oo was determined 2 hr after an accident while the patient was still under the effects of evipan (hexobarbitone). In court it was claimed that evipan had caused the high blood alcohol value. In an experiment, blood samples were taken from 7 persons 1 hr before, during, and 1 hr after awakening from evipan narcosis. The values are tabulated, and show that evipan had no effect on the blood alcohol levels.

1456. Werner, H. W.

CERTAIN EFFECTS OF BENZEDRINE, CORAMINE, METRAZOL AND PICROTOXIN IN ALCOHOL DEPRESSION.

J. Pharmacol. Exp. Ther. (Baltimore), 66: 39 (0 ref.),

1939.

E – abst. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – mot. perform. – CNS – amphetamines – stimulants – *CAAAL-0 A-1218.

The results of this investigation indicate that the administration of 7 cc/kg of alcohol po to rabbits provides protection against the lethal effects of subsequently administered benzedrine, coramine, metrazol, or picrotoxin. The lethal dose of benzedrine was increased approximately 1.5 times, and the lethal dose of each of the other 3 stimulants was increased approximately 2 times. Some evidence of stimulation was noted after the use of each of the analeptics, but none effected complete arousal of depressed animals.

1457. Werner, H. W.

THE EFFECTS OF BENZEDRINE, CORAMINE, METRAZOL AND PICROTOXIN ON BODY TEMPERATURE AND GASEOUS METABOLISM IN RABBITS DEPRESSED BY ALCOHOL.

J. Pharmacol. Exp. Ther. (Baltimore), 72: 45 (0 ref.),

1941.

E – abst. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – CNS – metab. proc. – amphetamines – stimulants – *CAAAL-3238-D2 A-1219.

The effects of analeptics in antagonizing the depression of body temperature induced by ethanol were investigated in rabbits. Determinations at approximately 1 to 3 hr after giving analeptics (about 5 to 7 hr after alcohol) showed that benzedrine and coramine were most effective in increasing body temperature, while benzedrine and metrazol most consistently increased metabolic rate at this time. At about 5 to 7 hr after the analeptics, rectal temperature and metabolic rates were usually near preanaleptic levels, but increased metabolic rates were observed in some experiments with each of the analeptics.

1458. Weyrich, G.

- ÜBER DAS ANTIALKOHOLMITTEL „PROMILL-EX“. [On the antialcoholic drug Promill-EX].

Ärztliche Mitteilungen (Cologne), 44: 231-232 (0 ref.),

1959.

G – exp. – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – mot. perform. – psychol. perform. – CNS – *CAAAL-0 A-1220.

Promill-EX contains catalytically-acting ferment systems from yeast, lecithin, and caffeine. Human subjects ingested 6 tablets of the drug plus 240 cc cognac within 1 hr, and then underwent psychological tests, blood tests, and clinical observation. Promill-EX did not influence the blood alcohol level, and had no effect on impairment caused by alcohol.

1459. White, R. L.

THE COMBINED USE OF INTRAVENOUS ETHYL ALCOHOL AND INTRAVENOUS PITOCIN.

Amer. J. Obstet. Gynec. (St. Louis), 63: 854-855 (12 ref.),

1952.

E – exp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – CNS – respir. – skel., muscle, skin – analg., antipyret. – autonomic agents – barbiturates – elect., water-bal. agents – hormones, hormone antag. – *CAAAL-6224-V35 A-1221.

A sol of 7.5% ethanol in 5% dextrose in water was combined with a pitocin sol (1 oxytocic unit of pitocin to 50 cc 5% dextrose in water) through a guage needle attached to a Y tube. The combined

use of pitocin and alcohol seemed to have a synergistic effect in patients in labour. Additional medication in the form of demerol and scopolamine was given if the patient requested it. The dosages of demerol and scopolamine were decreased to 1/3 or 1/4 of what had been used prior to the use of alcohol iv. The clinical sedative response to demerol was found to be greatly increased when an adequate amount of alcohol had been given.

1460. White, R. L.

INTRAVENOUS ETHYL ALCOHOL ANALGESIA WITH INTRAVENOUS PITOCIN
INDUCTION OF LABOR.

Amer. J. Obstet. Gynec. (St. Louis), 70(5): 983-986 (18 ref.), 1955.
E – exp. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – CNS – respir. – skel., muscle, skin – analg., antipyret. – autonomic agents – elect., water-bal. agents – hormones, hormone antag. – *CAAAL-7029-V35 A-1222.

Anesthesia with an iv combination of pitocin (5 oxytocic units, added to 1000 cc 5% dextrose in water) and 95% alcohol (90 cc, added to 910 cc 5% dextrose in water) was administered to 250 women during labour. No contraindications were found to the anesthesia, and there was no inconvenience in using iv alcohol at the same time as iv pitocin. Patients required a minimal amount of watching during labour, because any excessive amount of alcohol was easily noted and controlled. The effect of the iv pitocin and the conduct of the labour and delivery were not affected by the iv alcohol. Very minimal amounts of demerol and scopolamine were necessary to produce deep sedation and amnesia for the mother. All of the babies cried spontaneously, and showed no evidence of respiratory depression.

1461. Whitney, D. D.

THE POISONOUS EFFECTS OF ALCOHOLIC BEVERAGES NOT PROPORTIONAL TO
THEIR ALCOHOLIC CONTENTS.

Science (Washington), 33(850): 587-590 (O ref.), 1911.
E – exp. comp. – congen. stud. – other org. – acute admin. – in vivo – *CAAAL-0 A-1358.

The author attempted to show that the 3 main kinds of alcoholic beverages, i.e., wines, malt beverages, such as ales and beers, and distilled beverages, such as whisky, gin, and brandy, contain other important toxic ingredients besides ethanol, and to demonstrate that various alcoholic liquors, when reduced to the same percentage of alcohol, differ widely in their toxicity. *Hydantina senta* (a species of rotifer) were placed in mixtures of 1 cc foul water containing protozoa and 9 cc tap water; varying amounts of different alcoholic beverages were added, and 2 indicators of toxicity noted: percentage alcohol present in diluted beverage at which animals died within 10-30 min, and highest percentage alcohol present in diluted beverage at which females produced apparently normal young. It was found that sherry and port wine, containing 20 parts of alcohol, were much more toxic than beer and ale, and Holland gin, containing about 50 parts of alcohol, was much less toxic than any beer or wine (ranging from 3 to 30 parts alcohol). Another experiment illustrated that alcohol-free claret and sherry retained some toxicity upon rotifers, and, as such, each beverage was more toxic than absolute alcohol. Wines are considered to be the most toxic, followed by malt beverages, and, lastly, distilled liquors (which most closely approach absolute alcohol toxicity).

1462. Whittlesey, P.

THE EFFECTS OF PENTOBARBITAL ON THE METABOLISM OF ETHYL ALCOHOL
IN DOGS.

Johns Hopkins Hospital, Bulletin (Baltimore), 95: 81-89 (12 ref.), 1954.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – liver, kidney – metab. proc. – respir. – barbiturates – *CAAAL-7142-A2 A-1223.

The effect of pentobarbital anesthesia on the rate of disappearance of alcohol from the serum of dogs was studied in 44 experiments with 4 dogs. Following pentobarbital anesthesia (25 mg/kg in a 2.5% aqueous sol iv), 10 g/kg of a 10% alcohol sol was given iv over a 5-8 min period. The results showed that pentobarbital anesthesia in the above doses effects a reduction in the rate of decrease of serum alcohol averaging between 4 and 18%. The day to day variability in the rate of decrease of serum alcohol is reduced by pentobarbital, but the hour to hour variability in the rate is essentially unaltered. The rate of decrease of serum alcohol in unanesthetized dogs shows a slight but significant tendency to decrease with decreasing serum alcohol concentration. However, no significant departure from linearity could be ascertained under the influence of pentobarbital at the doses used, over the range of alcohol values obtained.

1463. Wiberg, G. S., Coldwell, B. B., and Trenholm, H. L.

TOXICITY OF ETHANOL-BARBITURATE MIXTURES.

J. Pharm. Pharmacol. (London), 21(4): 232-236 (17 ref.), 1969.

E – exp. cont. – exp. comp. – DC (supra-add. incr.) – mammals – acute admin. – in vivo – dose resp. – blood lev. – other drug lev. – absorp., distrib., stor. – CNS – metab. proc. – barbiturates – sed., hypnot. – *CAAAL-0 B-0489.

The simultaneous administration of ethanol at doses of either 2, 3, or 4 g/kg ip produced a dose-related decrease in the ip LD₅₀ for thiopentone, pentobarbitone, amylobarbitone, phenobarbitone, and barbitone in rats. The most marked ethanol-barbiturate interaction was with the long-acting, poorly-metabolized, less potent barbiturates, phenobarbitone and barbitone. Similarly, a non-hypnotic dose of ethanol (3 g/kg ip) produced a much greater prolongation of the sleeping time with non-hypnotic doses of phenobarbitone and barbitone, than with threshold doses of the shorter-acting barbiturates. Various postulates are advanced to explain the underlying mechanism of the barbiturate-ethanol interaction.

1464. Wiberg, G. S., Coldwell, B. B., and Trenholm, H. L.

TOXICITY OF ETHANOL BARBITURATE MIXTURES.

J. Pharm. Pharmacol. (London), 22(6): 465 (7 ref.), 1970.

E – exp. – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – blood lev. – CNS – liver, kidney – respir. – barbiturates – *CAAAL-0 B-0587.

In a reply to the criticism of S.E. Smith and A. Herxheimer (J. Pharm. Pharmacol., 21: 869-870, 1969) of the initial study of the authors on alcohol-barbiturate interaction (J. Pharm. Pharmacol., 21(4): 232-236, 1969), they contend that their experiments were not designed to establish whether such interaction is “additive” or “synergistic”. However, further research by the authors has established that: 1) 3 g/kg ethanol ip plus barbiturates markedly reduces blood pressure, and barbiturate urine formation and renal clearance is reduced; 2) ethanol reduces body temperature directly in proportion to the dosage (as much as 2-3°—an effect which would be expected to reduce barbiturate and ethanol hepatic metabolism; 3) ethanol lowers respiration rate and blood pO₂ levels—an effect which would be expected to decrease barbiturate and ethanol metabolism; 4) pentobarbitone increases purified rat liver dehydrogenase activity, but decreases the metabolism by liver slices; 5) the toxicity of ethanol and sensitivity to ethanol plus barbiturate are increased in older rats (12-14 months); and 6) ethanol alters the distribution of barbiturates in body compartments. The authors are convinced that use of the terms “additive” or “synergistic” does not help to clarify understanding of the combined effects of ethanol and barbiturates.

1465. Wiberg, G. S., Coldwell, B. B., Trenholm, H. L., and Thomas, B. H.

TOXICODYNAMIC STUDIES ON THE INTERACTION BETWEEN ETHANOL AND BARBITURATES.

Industr. Med. Surg. (Chicago), 39(7): 311 (0 ref.), 1970.

E – abst. – exp. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo – in vitro – blood lev. – other drug lev. – absorp., distrib., stor. – cardiovasc. – CNS – liver, kidney – metab. proc. – respir. – barbiturates – *CAAAL-0 B-1002.

The lethal doses and prolongation of sleeping times of mixtures of ethanol and 5 barbiturates were studied in rats. Greatest synergistic effects were obtained with long-acting, poorly-metabolizing barbiturates. Simultaneous administration of 3 g/kg ethanol ip altered the blood decay profile of phenobarbital. Other changes resulting from the drug combinations were diminished rates of urine formation and renal clearance of barbiturate, and decreases in body temperature, respiration rate, and blood pO₂ levels. Barbiturates did not appreciably alter the in vivo rate of ethanol metabolism. In vitro studies of barbiturate effects on ethanol metabolism were conducted on liver slices and purified liver alcohol dehydrogenase (ADH). Pentobarbital enhanced the activity of purified ADH, but retarded ethanol metabolism in liver slices. The effect of ethanol on the distribution of barbiturates was studied in 17 organs and tissues. Among several changes found was an enhanced barbiturate uptake by the brain.

1466. Widmark, E. M. P.

ÜBER DIE KONZENTRATION DES GENOSSENEN ALKOHOLS IN BLUT UND HARN UNTER VERSCHIEDENEN UMSTÄNDEN. [The concentration of ingested alcohol in blood and urine under various conditions].

Skandinavisches Archiv für Physiologie (Leipzig), 33: 85-96 (4 ref.), 1916.

G – SEC – exp. comp. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – anticonvulsants – sed., hypnot. – *CAAAL-3937-A1 A-1464.

A series of self experiments were performed by the author to study the correlation of alcohol concentrations in blood and urine under various conditions. In 1 experiment, the author (a 76 kg-male, accustomed to moderate alcohol use) ingested, on an empty stomach, 200 cc of a 10.9% alcohol sol containing 20 g of magnesium sulphate. The alcohol content of the urine was determined every 15 min for 90 min. 1/2 hr following ingestion, diarrhea occurred. The max alcohol concentration was 0.48°/oo, a level 22% lower than that obtained after ingestion of alcohol alone. It is concluded that magnesium sulphate, a salt which is difficult to absorb, has an inhibiting effect on alcohol absorption.

1467. Widmark, E. M. P.

ÜBER DIE EINWIRKUNG DER DINITROPHENOLE AUF DIE UMSETZUNGSGESCHWINDIGKEIT DES ÄTHYLALKOHOLS. [The action of dinitrophenols on the rate of oxidation of ethyl alcohol].

Biochemische Zeitschrift (Berlin), 276: 268-270 (2 ref.), 1935.

G – exp. comp. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – blood lev. – metab. proc. – respir. – skel., muscle, skin – indust. intox. – miscellaneous – *CAAAL-1122-A2 A-1224.

The effects of dinitrophenol and dinitrocresol upon the blood alcohol curve were investigated in dogs (wt 18.3 and 18 kg) after they had been given 31.6 g ethanol by stomach tube. 1,2,4-dinitrophenol and o-dinitrocresol after oral application markedly increased the rate of disappearance of alcohol from the blood. The author believes that this phenomenon is produced by increased metabolism, and not by increased temperature or increased respiration. 1,2,5- and 1,2,6-dinitrophenol did not influence the blood alcohol curve.

1468. Wier, J. K., and Tyler, V. E., Jr.

AN INVESTIGATION OF *COPRINUS ATRAMENTARIUS* FOR THE PRESENCE OF DISULFIRAM.

J. Amer. Pharm. Ass. (Washington), 49(7): 426-429 (20 ref.),

E – exp. – DC (sensit.) – unclass. ther. agents – *CAAAL-0

1960.

A-1359.

The authors investigated the contention by Simandl and Franc (Chemicke Listy, 50: 1862-1863, 1956) that disulfiram could be isolated from the mushroom *Coprinus atramentarius*. *Coprinus* cultures from Baarn, Holland were grown in 7 selected media, and concentrated extracts of media and *Coprinus* mycelia were analyzed by paper partition chromatography. Naturally-occurring carpophores from local *Coprinus* (recently reported as having produced alcohol intolerance) were similarly analyzed. In addition, disulfiram was added to the culture medium. Although the extraction process was 50% efficient, and could detect a 100 µg quantity of disulfiram, no disulfiram was found. For disulfiram to have been undetected, the culture media extracts must have contained less than 0.2 mg%, mycelium extracts less than 80 mg%, and extracts of naturally-occurring carpophores less than 0.4 mg%. At such low levels, 125 kg fresh mushrooms would be necessary to contain a normal maintenance dose of disulfiram (500 mg), but *Coprinus* from the same mycelial source possesses disulfiram-like activity when consumed in ordinary amounts, and, therefore, some other causative agent must exist. Added disulfiram had no observable effect on organism development, and disappeared within 60 days. Several possibilities might explain how Simandl and Franc found a recoverable concentration of disulfiram in their investigation: their "*C. atramentarius*" may not have been identical; nutritional and environmental factors altered the fungi metabolically, with resultant quantitative changes; or different genetic strains of *Coprinus* may exist which differ in their metabolism.

1469. Wikler, A.

SURVEY OF RESEARCH ON ALCOHOL AT THE NATIONAL INSTITUTE OF MENTAL HEALTH, ADDICTION RESEARCH CENTER, LEXINGTON, KY., U.S.A.

In: *Twenty-sixth International Congress on Alcohol and Alcoholism, Abstracts*. Stockholm, Sweden, August 1-5, pp. 20-23 (0 ref.), 1960.

E – abst. – presentation – cross-tol. – drug-dep. humans – chronic admin. – in vivo – blood lev. – other drug lev. – CNS – barbiturates – nutritive agents – *CAAAL-0 A-1225.

2 experiments were carried out, involving the effects of alcohol and other substances in adult male narcotic addicts. In the first experiment, the test subjects were continually maintained in a state of alcohol intoxication for 6-87 days, consuming 346-487 ml alcohol daily. They received a well-balanced high calorie diet, supplemented with vitamins A, B, C, and D. Upon sudden withdrawal of alcohol, the subjects suffered the usual symptoms, indicating that vitamin deficiency is not a necessary condition for delirium tremens. In the second experiment, subjects addicted to barbiturates were given alcohol (270-460 ml/day for 14 days) in place of barbiturates. There was a reduction in the incidence of barbiturate withdrawal phenomena. Upon abrupt withdrawal of alcohol, the abstinence syndrome that followed was more characteristic of alcohol than barbiturates.

1470. Wilbur, D. L., MacLean, A. R., and Allen, E. V.

CLINICAL OBSERVATIONS ON THE EFFECT ON BENZEDRINE SULFATE: A STUDY OF PATIENTS WITH STATES OF CHRONIC EXHAUSTION, DEPRESSION AND PSYCHONEUROSIS.

J.A.M.A. (Chicago), 109(8): 549-554 (19 ref.), 1937.

E – SEC – general – DC (decrease) – psychot. humans – CNS – amphetamines – *CAAAL-0 A-1226.

The effects of benzedrine sulphate on 100 patients suffering from chronic exhaustion, depression, and psychoneurosis were studied; the immediate results of oral administration (1-20 mg, twice daily) in the foregoing 3 conditions were that the drug was beneficial in 80%, 70%, and 46% of the cases, respectively. The results of the continued administration of benzedrine were less favourable. Individual cases are described. It is noted that, "some persons who have indulged in too large quantities of alcohol may find that the characteristic morning 'hangover' is greatly benefited by benzedrine."

1471. Wilkinson, P., Horvath, T. B., Santamaria, J. N., and Rankin, J. G.
BROMISM IN ASSOCIATION WITH ALCOHOLISM: A REPORT OF FIVE CASES.
 Med. J. Aust. (Sydney), 1(26): 1352-1355 (21 ref.), 1969.
 E – general – case hist. – conj. addict. – drug-dep. humans – blood lev. – CNS – sed., hypnot. –
 *CAAAL-13922 B-0588.

With 5% of all alcoholic patients, in the experience of the authors, using bromide preparations at some time or other, the possibility of habituation leading to chronic bromide intoxication is considerable, due to free non-prescriptive use of bromides to allay tensions and anxieties of alcohol withdrawal. Bromism may be mistaken for multiple sclerosis, since the symptoms are similar: nystagmus, slurred speech, ataxia, and toxic amblyopia. Bromide habituation can complicate or replace alcohol dependency, and can produce symptoms which may be mistaken for dementia due to alcohol. Patients who were alcoholics or recovered alcoholics were found taking up to 70 "seda-tabs" per day, and had serum bromide levels as high as 290 mg/100 ml, the therapeutic level being 75-125 mg/100 ml. These patients showed confusion, disorientation, slurred speech, visual problems, and psychological disturbance. Treatment was a combination of forced diuresis with mersalyl or chlorothiazide, fluid intake, and sodium chloride supplements, following which the serum level decreased, and toxic symptoms subsided.

1472. Willard, P. W., and Horvath, S. M.
CORONARY CIRCULATION DURING AND FOLLOWING ETHYL ALCOHOL INFUSION.
 Arch. Int. Pharmacodyn. (Gand), 148(1-2): 181-185 (6 ref.), 1964.
 E – SEC – exp. cont. – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – in vivo –
 blood lev. – cardiovasc. – barbiturates – *CAAAL-0 A-1450.

8 mongrel dogs were anesthetized with sodium pentobarbital (30 mg/kg) and heparinized (5 mg/kg), after which coronary blood flow and various parameters were calculated. A total of 500 mg 95% alcohol/kg was then infused into the saphenous vein over a 60 min period, following which the measurements were again taken. Coronary blood flow and cardiac output were determined 45 min after the start, and 20 min after the cessation, of infusion. The observation by other authors of decreased myocardial efficiency in dogs under barbiturate anesthesia was substantiated. During infusion, 2 animals showed a 68% increase over control values in coronary flow, 3 showed a 19% decrease, and the others exhibited no change; 20 min after the cessation of infusion, the increased coronary flow in 2 dogs remained elevated, with a 13% decrease from the infusion value, while the other animals showed no change from infusion values. Cardiac output decreased during infusion, and decreased significantly after infusion. Systolic and diastolic pressures decreased significantly, and pulse pressure was simultaneously lowered, reflecting a fall in stroke vol. A significant increase in total body oxygen consumption and carbon dioxide production was observed during infusion. Cardiac oxygen consumption increased, but heart work decreased, with a resulting loss in efficiency.

1473. Williams, M. B.
MANAGING ALCOHOLICS WITH SERPASIL AND RITALIN.
 Virginia Med. Monthly (Richmond), 88(5): 269-271 (5 ref.), 1961.
 E – general – DC (add., infra-add., unspec. incr.) – drug-dep. humans – dose resp. – mot. perform.
 – psychol. perform. – stimulants – tranquilizers – *CAAAL-9715-N47 A-1360.

The author investigated the value of reserpine and ritalin in treating 107 male alcoholics, aged 40-60, in a private sanatorium, in conjunction with other withdrawal procedures. Large doses of reserpine alone caused depression, although the patients slept better and required less alcohol and other sedation during the withdrawal period. The addition of the stimulant, ritalin, to the dosage schedule eliminated the depressing effects. Therapeutic doses of ritalin increased alertness and psychomotor activity in patients with fatigue, oversedation, and depression symptoms. The optimum dosage appeared to be

5 mg reserpine im upon admission, followed by 1 mg daily for 5 days, in conjunction with 20-60 mg daily of ritalin in divided doses, a treatment which was continued until discharge of the patient. Ritalin alone was quite effective in the depressed or remorseful patient, when used in addition to the routine withdrawal of alcohol. Previously difficult patients with a high tolerance to alcohol and other sedatives were given 50-100 mg promazine im the first night, in addition to the routine reserpine-ritalin schedule, and, in the presence of alcohol, the calming effect was dramatic. "A belligerent, noisy, and restless patient could be calmed down and usually put to sleep in a matter of minutes." Doriden was found to be the best all-round general night-time sedative, both with and without alcohol intoxication.

1474. Wilson, A. S., Barboriak, J. J., and Kass, W. A.

EFFECTS OF ALCOHOLIC BEVERAGES AND CONGENERS ON PSYCHOMOTOR SKILLS IN OLD AND YOUNG SUBJECTS.

Quart. J. Stud. Alcohol (New Haven), Suppl. No. 5: 115-129 (11 ref.), 1970.
 E – exp. cont. – congen. stud. – humans – acute admin. – in vivo – mot. perform. – psychol. perform.
 – CNS – *CAAAL-12893 B-0589.

The effects of alcohol on different age groups were determined, and the effects of vodka and bourbon were compared. 2 groups of 30 men each, 1 group containing 60-85 yr-old subjects, and the other 21-35 yr-old men, were randomly served with vodka or bourbon (0.75 g/kg alcohol content), or water, each person serving as his own control. Digit symbol, hand steadiness, and body sway tests were made before, and 1/2 hr after, dose administration. Although it was expected that bourbon, with its high congener content, would cause greater impairment than vodka, which is low in congeners, the results were ambiguous. Vodka significantly influenced the performance of the older group in the number of digit symbols attempted and the number of body sways, while bourbon did not. On the other hand, bourbon significantly affected the performance of the older group in the number of hand steadiness hits, whereas vodka did not. It is conjectured that moderate amounts of ethanol may facilitate certain forms of behavior, by affecting certain behavioural variables associated with a given test, through indirect action on the variables, rather than by directly affecting measured behaviour; hence, bourbon might produce a depressant effect beyond that which could favourably influence the proprioceptive motor behaviour in body sway, and thus reduce performance, whereas the different sensitivity of functional elements involved in hand steadiness, as well as different secondary variables, may respond favourably to bourbon, and result in improved performance.

1475. Wilson, A. S., Barboriak, J. J., and Lech, C. C.

MODIFICATION OF ETHANOL EFFECTS BY CHLORCYCLIZINE IN THE RAT.

Proc. Soc. Exp. Biol. Med. (New York), 134(4): 993-995 (13 ref.), 1970.
 E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – mammals – chronic admin. –
 in vivo – blood lev. – liver, kidney – metab. proc. – unclass. ther. agents – *CAAAL-15218
 B-1032.

To determine whether the reported decrease in liquid intake by rats given dilute alcohol is due to a natural aversion to alcohol or to a temporary overloading in the capacity to metabolize alcohol, the effects of chlorcyclizine (a known inducer of drug-metabolizing enzymes) on chronic alcohol consumption and on hepatic triglyceride levels were investigated. 40 male rats (225-260 g) were divided into 4 groups. As the only source of liquid, the first group received tap water, the second a 15% w/v alcohol sol, the third tap water containing 12.5 mg chlorcyclizine hydrochloride/kg/day, and the fourth alcohol + chlorcyclizine (above doses). After 31 days, blood samples were drawn for lipid analysis, and livers were assayed for hepatic triglycerides. Ethanol significantly reduced total liquid intake, as did chlorcyclizine; when the 2 agents were given in combination, the ethanol-induced liquid intake reduction was significantly less pronounced. Ethanol did not affect hepatic triglyceride levels, whereas chlorcyclizine produced a slight increase; in rats given the combined dosage, a pronounced increase in liver triglycerides was observed. It is suggested that chlorcyclizine-enhanced ethanol

consumption may have contributed to the increased liver triglyceride levels, and thus it is concluded that chronic chlorcyclizine administration increases tolerance to alcohol.

1476. Wilson, L., Taylor, J. D., Nash, C. W., and Cameron, D. F.
 THE COMBINED EFFECTS OF ETHANOL AND AMPHETAMINE SULFATE ON
 PERFORMANCE OF HUMAN SUBJECTS.
 Canad. Med. Ass. J. (Toronto), 94(10): 478-484 (17 ref.), 1966.
 E – FS – exp. cont. – DC (decrease) – humans – acute admin. – in vivo – blood lev. – other drug
 lev. – mot. perform. – psychol. perform. – CNS – amphetamines – nutritive agents –
 *CAAAL-11333-J1 B-0214.

25 human subjects received ethanol (1.2 g/kg) plus amphetamine (15 g) or ethanol plus placebo, and performance on the balance, skipping, mental addition, coding, pursuit rotor, Minnesota rate of manipulation, Purdue peg board, digit span, trail-making, Bender-Gestalt, Wonderlic Personnel Test, and Maudsley Personality Inventory was evaluated. The interrelationships between ethanol and amphetamine were found to be complex. No differences between the combined drug dose and the control dose were found for the balance, skipping, Minnesota manipulation, Purdue peg board, Maudsley Personality Inventory, pursuit rotor, and digit span tests. Ethanol-amphetamine produced less impairment of performance in the coding, mental addition, and trail-making tests than ethanol-placebo. Ethanol increased error on the Wonderlic Personnel test, and ethanol-amphetamine failed to improve performance; however, amphetamine reduced the test-retest reliability of the test, and ethanol was found to counteract the amphetamine effect. It is concluded that each drug modified some of the effects of the other, but the combined effects cannot be predicted on the assumption that a depressant versus stimulant competition is operative.

1477. Wilson, R. H.
 DIAGNOSIS AND TREATMENT OF INDUSTRIAL SOLVENT POISONING.
 J.A.M.A. (Chicago), 139(14): 906-909 (0 ref.), 1949.
 E – SEC – general – DC (add., infra-add., unspec. incr.) – humans – miscellaneous –
 *CAAAL-5116-D3 A-1227.

Poisoning with aromatic hydrocarbons, chlorinated hydrocarbons, ketones, alcohols, petroleum distillates, and carbon disulfide is discussed. Treatments are outlined for each of the above substances. It is emphasized that persons exposed to carbon disulfide should abstain from alcohol.

1478. Winfield, D. L.
 THE USE OF METHYPRYLON AS AN AID IN OBTAINING
 ELECTROENCEPHALOGRAMS IN ALCOHOLICS.
 J. Nerv. Ment. Dis. (Baltimore), 130(1): 45-48 (14 ref.), 1960.
 E – exp. – DC (unchanged) – drug-dep. humans – acute admin. – in vivo – cardiovasc. – CNS – sed.,
 hypnot. – *CAAAL-9325-E8 A-1228.

Electroencephalographic changes and length of time for induction of sleep were studied in 82 patients, of whom 75 were alcoholics, 1 was a drug addict, and 6 were both. The subjects received an initial dose of 600 mg methyprylone, followed by another 400 mg if the subjects did not appear sleepy, or 800 mg followed by an additional 400 mg. Satisfactory EEG readings were obtained on all patients. No untoward effects due to the administration of methyprylone were noted. Methyprylone did not appear to potentiate or enhance the alcoholic state, and there was no apparent depression of blood pressure or pulse rate following administration.

1479. Wismer, P. -H.

ZUR FRAGE DES EINFLUSSES DER ALKOHOLGEWÖHNUNG AUF DIE WIRKUNG VON LOKALANALGETICA. [On the question of the influence of alcohol habituation on the effect of local analgetics].

Dissertation, Medical Faculty of the University of Hamburg, West Germany, 55 pp. (33 ref.),

1962.

G – exp. comp. – cross-tol. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – CNS – metab. proc. – anesthetics – integ. syst. agents – *CAAAL-0

A-1229.

Experiments showed that, over a 6-8 week trial period, guinea pigs adapted readily to a 5-20% alcohol sol as their sole liquid intake; the total daily intake of alcohol was 1.13 g/day in the 5% group, 1.66 g/day in the 10% group, 1.97 g/day in the 15% group, and 2.089 g/day in the 20% group. The local analgetics used were: 1) procaine, tetracaine, lidocaine, carbocaine, L 67, and baycaine, and 2) polyethoxydodecan. All local analgetics were used in 0.1% sol, except tetracaine, which was used in 0.01% sol. In guinea pigs habituated to alcohol, the duration of anesthesia was shortened with substances in group 1) and was increased with polyethoxydodecan. It is hypothesized that alcohol changes the sensibility of the nervous structure, but does not influence the metabolism of the local analgetics.

1480. Wójcicki, J.

WPŁYW PRZEWLEKŁEGO ALKOHOLIZMU DOŚWIADCZALNEGO NA REAKTYWNOŚĆ WYOSOBNIONEGO SERCA SZCZURA NA STROFANTYNĘ I TRUCIZNY UKŁADU WEGETATYWNEGO. [The influence of protracted experimental alcoholism on the reactivity of the isolated rat heart to strophanthin and poisons of the vegetative system].

Dissertationes Pharmaceuticae et Pharmacologicae (Warsaw), 19: 9-14 (16 ref.),

1967.

Po – ES – RS – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – mammals – acute admin. – chronic admin. – in vitro – acid-base, blood pH, elect. – cardiovasc. – skel., muscle, skin – stimulants – *CAAAL-0

B-1003.

Experiments were performed to study the effects of prolonged ethanol consumption on the reaction of isolated cardiac muscle of the isolated mouse heart to strophanthin, adrenalin, and acetylcholine. 12 male white mice (160-210 g) were divided into 2 groups. For 70 days, 1 group received saline, and the other received 1.5 ml/100 g 20% ethanol sol/day by gastric tube. Then the inotropic and chronotropic reaction of the heart to 10 µg/0.1 ml strophanthin, 1 µg/0.1 ml adrenalin, or 100 µg/0.1 ml acetylcholine were determined by the Langendorff method. In the hearts of rats given ethanol chronically, the effect of strophanthin on heart muscle contractility was increased, and that of adrenalin and acetylcholine was decreased. The change in heart rate induced by strophanthin was unaffected, whereas the reaction to adrenalin was increased, and that to acetylcholine was decreased. It is concluded that prolonged ethanol ingestion can influence the exchange of ions (especially of potassium), and that this in turn affects heart muscle reactivity.

1481. Wolf, M.

UNTERSUCHUNGEN ÜBER ERNÜCHTERUNGSMITTEL. [Investigations of sobering-up drugs].

Dissertation, Medical Faculty of the University of Heidelberg, West Germany, 92 pp. + 29 graphs (43 ref.),

1960.

G – exp. – DC (decrease) – DC (unchanged) – humans – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – metab. proc. – analg., antipyret. – anti-infectants – barbiturates – elect., water-bal. agents – tranquilizers – *CAAAL-0

A-1230.

19 alcohol tests were carried out on male and female subjects to determine the effect of "bacchantyn" and "promill-EX" as sobering agents. The composition of the former is unstated; the latter contains catalytically-acting ferment systems from yeast, plus lecithin and caffeine. Different drinks were ingested—300 cc cognac (31 g alcohol/100 cc), 750 cc champagne (8.75 g alcohol/100 cc), and beer (3.87 g alcohol/100 cc). Blood alcohol levels were determined by the Widmark method, and were found to range from 0.75 to 1.65°/oo. The absorption time in the alcohol tests was 20-50 min. In 1 case, absorption was completed after 5 min, and in another after 160-170 min. 9 of the 19 blood alcohol curves appeared normal, and 10 irregular. It is concluded that neither agent had a significant effect on the degree of intoxication or on subjective feeling.

1482. Wolff, H. G., Hardy, J. D., and Goodell, H.
STUDIES ON PAIN: AN ANALYSIS OF THE ANALGESIC ACTION OF ETHYL ALCOHOL.

Trans. Ass. Amer. Physicians (Philadelphia), 56: 317-319 (1 ref.), 1941.
E – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – skel., muscle, skin – analg., antipyret. – *CAAAL-3516-D1 A-1231.

The pain threshold was studied in 3 human subjects. The amount of heat sufficient to cause a painful sensation was determined as the pain threshold. Max threshold-raising effect was produced by 30 cc of 95% alcohol, 45% more heat being required to produce the pain sensation. The same amount of alcohol with 0.3 g acetylsalicylic acid did not elevate the threshold above 45%, but caused the effect to persist for 4 hr, and the max threshold was maintained for 90 min. The speed of onset of threshold-raising action and the changed psychological state after alcohol, coupled with the longer duration of the threshold-raising effect of the acetylsalicylic acid, make this a desirable combination of analgesic agents.

1483. Wölkart, N.
IST DIE VERABREICHUNG VON PARALDEHYD BEI ALKOHOLIKERN KONTRAINDIZIERT? [Is the administration of paraldehyde contraindicated in alcoholics?].

Wiener Archiv für Psychologie, Psychiatrie und Neurologie (Vienna), 4: 145-148 (20 ref.), 1954.
G – general – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – absorp., distrib., stor. – cardiovasc. – CNS – liver, kidney – metab. proc. – respir. – sed., hypnot. – *CAAAL-7250-N16 A-1232.

The author points out that it is conceivable that the toxicity of paraldehyde may be potentiated by alcohol or alcoholism. Paraldehyde is a cortical hypnotic—the absorption is fast, and elimination is slow. It is oxidized by the liver; hence, in cases of liver damage, the portion eliminated through the lungs is greater. In acute intoxication, the accumulation of acetaldehyde is possible; in the chronic condition, a degenerative effect of alcoholism on the cortex may effect a diminished resistance of the organ which is affected by paraldehyde. 2 cases of death due to paraldehyde poisoning are reported. A 25 yr-old chronic alcoholic received 4 doses of 10 g each of paraldehyde within 5 hr, and a 56 yr-old man, acutely intoxicated, received 10 cc of paraldehyde. The blood alcohol level of the former was 0.25°/oo, and that of the latter was 2.8°/oo. Both died suddenly—obviously of circulatory failure, and not of respiratory paralysis.

1484. Wood, C. A., and Buller, F.
POISONING BY WOOD ALCOHOL: CASES OF DEATH AND BLINDNESS FROM COLUMBIAN SPIRITS AND OTHER METHYLATED PREPARATIONS.

J.A.M.A. (Chicago), 43: 972-977, 1058-1062, 1117-1123, 1213-1221, and 1289-1296 (54 ref.), 1904.
E – SEC – general – case hist. – DC (antidotal) – humans – alcohols – anti-infectants – stimulants – *CAAAL-0 A-1233.

Tabulated records of 235 (published (54) and unpublished (181)) cases of methanol poisoning are presented. The pathology, symptoms, and treatment of the condition are discussed. Mention is made of 5 cases (3 fatal) treated by "Assistant Surgeon R." of the United States Army. One patient who completely recovered received 1 dose of 44 cc oleum ricini, followed by 30 cc whiskey/hr for an unstated length of time. The second survivor, partially blinded, initially received 1/20 grain strychnine sulphate and 8 cc whiskey hypodermically, followed by a further dose of strychnine and whiskey (dosages not stated; period of time not stated). The 3 fatal cases presumably also received ethanol. It is added that "Dr. R." is of the opinion, based on this experience, that, "methyl must be replaced by ethyl alcohol, in order to combat a collapse and sustain the patient's vitality. This, with the speedy clearing out of the poison from the alimentary canal and the continued stimulating and supportive treatment for several weeks, should constitute the principal aim in treatment."

1485. Woodhouse, S. W.

ON THE BITE OF THE RATTLESNAKE.

Buffalo Medical Journal and Monthly Review of Medical and Surgical Science (Buffalo), 8: 72-75 (0 ref.), 1852.

E – general – case hist. – DC (antidotal) – humans – stimulants – *CAAAL-0

A-1234.

The author describes self-treatment for snake bite, in which both ammonia and alcohol were taken internally. Upon being advised to take the *Western Remedy* (i.e., get drunk), of which the author had often heard, half a pint of whiskey was taken almost immediately. When camp was reached, more careful treatment followed, and the increased tolerance to alcohol was noted. "[I] then commenced drinking brandy, at the same time held my finger in a cup of ammonia. It took one quart of fourth proof brandy and half a pint of whiskey (enough to have killed a man under ordinary circumstances) to produce intoxication, which only lasted about four hours." Ammonia and cathartics were also taken, and external treatment applied. A successful recovery was made, although the author's arm was in a sling for 2 months, and the finger that was bitten was deformed. A number of other instances of rattlesnake bite have come to the attention of the author, in which successful recovery was made if alcoholic intoxication was induced. In 1 case, a man bitten by a snake was given "nothing but whiskey" by a doctor, and, in 3 days, was fully recovered; 3 pints of whiskey were necessary to produce intoxication.

1486. Wooles, W. R.

PREVENTION OF THE ACUTE ETHANOL-INDUCED FATTY LIVER BY ANTIHISTAMINES AND STIMULANTS OF HEPATIC MICROSOMAL ENZYME ACTIVITY.

Toxic. Appl. Pharmacol. (New York), 12: 186-193 (15 ref.),

1968.

E – exp. cont. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – absorp., distrib., stor. – CNS – liver, kidney – metab. proc. – autotox. – sed., hypnot. – *CAAAL-0

B-0490.

Female rats were given promethazine, chlorcyclizine, brompheniramine, or diphenhydramine; all administrations were ip, 25 mg/kg for 3 days, or 25 mg/kg 6 hr prior to and 12.5 mg/kg 2 hr prior to, 3 or 6 g/kg ethanol as a 50% sol by oral intubation. Of the drugs tested, only chlorcyclizine, promethazine, and SKF 525-A were effective. Promethazine was effective when administered shortly prior to intubation, and chlorcyclizine and SKF 525-A were effective when administered daily for 3 days prior to intubation of ethanol. In addition to reducing the severity of the fatty liver produced by 6 g/kg of ethanol, chlorcyclizine completely prevented the fatty liver following 6 g/kg ethanol, and the drug-treated rats manifested lower blood alcohol concentrations than the control group throughout the first 4 hr after intubation with 3 g/kg. Chlorcyclizine contains a benzyl carbon atom, and SKF 525-A does not. Since both drugs were of similar effectiveness in reducing the severity of the fatty liver, it appeared that the protective effect of these agents was not related to their ability

to act as scavengers of free radicals, but was more likely related to their ability to stimulate the activity of hepatic microsomal enzymes.

1487. Wooles, W. R., and Weymouth, R. J.
 PREVENTION OF THE ETHANOL-INDUCED FATTY LIVER BY
 CHLORCYCLIZINE-INDUCED MAINTENANCE OF HEPATIC LIPID OXIDATION.
 Lab. Invest. (New York), 18(6): 709-714 (22 ref.), 1968.
 E – exp. cont. – DC (decrease) – mammals – acute admin. – in vivo – blood lev. – liver, kidney –
 metab. proc. – autocoids – *CAAAL-0 B-0491.

The time sequence of the development of the acute ethanol-induced fatty liver and the protective effect of chlorcyclizine were studied biochemically and histologically. Fatty liver produced by 3 mg/kg ethanol was observed 8 and 16 hr after intubation, and was indicated by a 117% and 87% increase in liver triglyceride levels above the values observed in the isocaloric glucose control group. Rats pretreated with chlorcyclizine (25 mg/kg daily for 3 days) and receiving the same dose of ethanol demonstrated neither biochemical nor histological evidence of fatty degeneration induced by ethanol.

1488. Wooles, W. R.
 DRUG PROTECTION AGAINST ACUTE ETHANOL INTOXICATION AND FATTY
 LIVER PRODUCTION.
 Industr. Med. Surg. (Chicago), 39(7): 326 (0 ref.), 1970.
 E – abst. – exp. comp. – DC (decrease) – mammals – acute admin. – in vivo – in vitro – blood lev.
 – liver, kidney – metab. proc. – *CAAAL-0 B-1004.

The protective effect of chlorcyclizine against acute ethanol intoxication and fatty liver production was evaluated in rats, using the ability to stimulate hepatic microsomal enzyme activity as a criterion. Chlorcyclizine did not alter mobilization of peripheral fatty acids produced by ethanol, but did maintain a normal rate of hepatic oxidation of fatty acids (this rate being depressed in rats given ethanol alone). Electron microscope studies revealed that ethanol markedly altered the size and shape of liver mitochondria, and that this effect was prevented by chlorcyclizine pretreatment. Ethanol-treated animals pretreated with chlorcyclizine showed a marked proliferation of smooth endoplasmic reticulum. Blood alcohol levels were also lower in drug-treated rats. The effect of drug pretreatment upon liver alcohol dehydrogenase (ADH) activity and the hepatic NAD/NADH₂ ratio was also studied. Ethanol decreased hepatic ADH activity, and reduced the liver NAD/NADH₂ ratio; both effects were offset by chlorcyclizine. It is suggested that the initial metabolic derangements produced by acute ethanol intoxication may be due to an alteration of the liver NAD/NADH₂ ratio, and that the protective effect of chlorcyclizine is at the level of hepatic nucleotides.

1489. Wright, F.
 CORAMINE IN ALCOHOLIC COMA.
 Clinical Medicine and Surgery (Waukegan), 47: 230 (0 ref.), 1940.
 E – general – DC (decrease) – humans – CNS – metab. proc. – stimulants – *CAAAL-2684-N13
 A-1235.

Clinical experience demonstrated that patients in alcoholic coma can be promptly restored to consciousness by an iv injection of from 1 to 15 cc of coramine, the dose depending on the condition of the patient. It is believed that the effect of the drug is due to an increase in tissue oxidation produced by this drug.

1490. Wuermeling, H. B., Leithoff, H., and Weyrich, G.
 UNTERSUCHUNGEN ÜBER EIN ANGEBLICH DIE
 BLUTALKOHOLKONZENTRATION SENKENDES MITTEL (PROMILL EX).

[Investigations of a drug purported to decrease the alcohol concentration in the blood (Promill EX)].

Med. Welt (Stuttgart), 41: 1935-1938 (2 ref.),

1959.

G – exp. cont. – DC (unchanged) – humans – blood lev. – *CAAAL-9341-A1

A-1236.

Promill EX (containing catalytically-acting ferment systems from yeast, plus lecithin and caffeine) or placebo capsules were administered together with 240 cc of cognac, to 14 subjects, and the blood alcohol levels determined. The blood alcohol levels (0.92-1.51°/oo) indicated that the drug is not capable of lowering blood alcohol levels, nor is it concluded that it is capable of mitigating the effects of alcohol.

1491. Yamamoto, R. S., Korzis, J., and Weisburger, J. H.

CHRONIC ETHANOL INGESTION AND THE HEPATOCARCINOGENICITY OF N-HYDROXY-N-2-FLUORENYLACETAMIDE.

Int. J. Cancer (Copenhagen), 22: 337-343 (37 ref.),

1967.

E – FS – exp. cont. – DC (decrease) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – chronic admin. – in vivo – species or sex diff. – liver, kidney – *CAAAL-0 B-0492.

2 series of tests were performed to study the effect of long-term administration of 10% and 20% ethanol sol on the hepatocarcinogenicity of N-hydroxy-N-2-fluorenylacetamide in 2 strains of rats. In tests with ethanol and the carcinogen, the size of the liver and the extent of tumor formation were slightly less with male, and slightly more with female rats, compared to rats fed the carcinogen only. Experiments beginning with 16 week-old males, or with ethanol following the carcinogen, were without additional effect. In NIH black rats, less susceptible to the carcinogen and with a longer experimental period, the concurrent intake of 10% ethanol did not affect the liver tumor incidence, although the female rats showed fewer cholangiomas and more hepatomas.

1492. Zaffiri, O., and Francescato, F.

PROBLEMI ANESTESIOLOGICI NELL'ALCOOLISMO ACUTO. [Anesthesiological problems in acute alcoholism].

Minerva Anest. (Turin), 33: 263-265 (7 ref.),

1967.

I – General – DC (add., infra-add., unspec. incr.) – DC (unchanged) – humans – drug-dep. humans – cardiovasc. – CNS – G.I. tract – liver, kidney – respir. – analg., antipyret. – anesthetics – antidepressants – antispasmodics – barbiturates – nutritive agents – sed., hypnot. – *CAAAL-0

B-1033.

The pharmacology and organic metabolism of alcohol, the physiopathology of acute alcoholic intoxication, anesthesiological conduct, and intra- and post-operative reanimation are discussed. For pre-anesthesia, iv chlorpromazine (25-50 mg) plus promethazine (50 mg iv) are preferred. Small doses of atropine are useful, and good results can also be obtained with diazepam and benzquinamide. Opiates and cortical sedatives (barbiturates) are contraindicated. In alcoholic coma, however, any preanesthetic, except atropine, is contraindicated. For anesthesia, local anesthetics are recommended wherever possible. Regarding general anesthesia, acute alcoholics usually require lower, or, at most, normal anesthetic doses, whereas chronic alcoholics are extremely resistant to anesthesia. A single iv dose of a thiobarbiturate (25-40 cg) is suggested; only in cases of violent psychomotor agitation should a larger dose be employed. The narcosis is best maintained by fluothane (0.5-1%), with a flow of 4 litres of a 50% oxygen-nitrous oxide mixture. Prior to or during anesthesia, anti-shock therapy (plasma expanders, and, later, blood transfusions) should be commenced. Vitamin B₆ (250-500 mg) and magnesium are helpful in intra- and post-operative reanimation. Electrolyte balance must be carefully followed. 1.4% sodium bicarbonate perfusions will overcome acidosis, and slow 5% mannitol perfusions will aid diuresis.

1493. Zakrividoroga, S. P.
SMESHANNOE I KOMBINIROVANNOE DEISTVIE NEKOTORYKH BARBITURATOV (MEDINALA I GEKSENALA) S KHLOROFORMOM, EFIROM I ALKOGOLEM NA IZOLIROVANNOE SERDTSE LIAGUSHKI. [Mixed and combined action of some barbiturates (medinal and hexenal) with chloroform, ether, and alcohol on the isolated heart of the frog].
Farmakol. Toksik. (Moscow), 9(1): 53-58 (14 ref.), 1946.
R – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (decrease) – other org. – in vitro – acid-base, blood pH, elect. – cardiovasc. – skel., muscle, skin – barbiturates – *CAAAL-0 A-1237.
- The effects of chloroform, ether, and alcohol, combined with barbital or hexobarbital, on changes in rhythm, amplitude, and tonus, were determined in 112 isolated frog hearts. It was found that hexobarbital and barbital mixed with chloroform, ether, and ethanol cause a summation of effects in therapeutic as well as in toxic concentrations. In their combined action, the toxicity of chloroform is clearer after the administration of hexobarbital, and the latter is more toxic after the administration of ether than after the administration of ethyl alcohol. The toxicity of hexobarbital is less strongly expressed subsequent to the action of chloroform, and the toxicity of ether and ethyl alcohol is decreased by the administration of hexobarbital.
1494. Zampi, G., and Smorlesi, L.
CELLULE MUCIPARE DEL TUBO GASTRO-ENTERICO E RIGENERAZIONE GASTRICA NEL BLOCCO FARMACODINAMICO SPERIMENTALE DEL SISTEMA NERVOSO AUTONOMO. [Muciparous cells of the gastroenteric tube and gastric regeneration in experimental pharmacodynamic block of the autonomic nervous system].
Arch. de Vecchi Anat. Pat. (Florence), 24: 235-262 (38 ref.), 1956.
I – exp. cont. – DC (decrease) – DC (unchanged) – mammals – acute admin. – in vivo – CNS – G.I. tract – nerv. syst. – *CAAAL-8701-D2 A-1238.
- Alcohol was introduced in 60% dilution directly into the stomach of guinea pigs whose autonomic nervous system was blocked by means of neuroplegic drugs. The production of mucus was increased by the alcohol, both in the presence and in the absence of the block.
1495. Zatman, L. J.
THE EFFECT OF ETHANOL ON THE METABOLISM OF METHANOL IN MAN.
Biochem. J. (London), 40: lxvii-lxviii (7 ref.), 1946.
E – abst. – exp. – DC (decrease) – humans – mammals – acute admin. – in vivo – in vitro – other drug lev. – liver, kidney – respir. – alcohols – *CAAAL-4748-A1 A-1239.
- In in vitro experiments with alcohol dehydrogenase, no detectable oxidation of methanol was observed when ethanol was present in equimolar concentration. Inhibition of methanol oxidation was demonstrable when the molar ratio of ethanol to methanol was only about 1 to 16, and the inhibition was found to be of the competitive type. This inhibitory effect was demonstrable in in vivo experiments on 3 human subjects; ethanol caused diminished oxidation of ingested methanol, and, therefore, increased excretion of methanol, both in the urine and in the expired air.
1496. Zbinden, G., Bagdon, R. E., Keith, E. F., Phillips, R. D., and Randall, L. O.
EXPERIMENTAL AND CLINICAL TOXICOLOGY OF CHLORDIAZEPOXIDE (LIBRIUM).
Toxic. Appl. Pharmacol. (New York), 3: 619-637 (15 ref.), 1961.
E – exp. cont. – exp. comp. – DC (add., infra-add., unspec. incr.) – humans – mammals – acute admin. – in vivo – CNS – antidepressants – sed., hypnot. – tranquilizers – *CAAAL-0 A-1240.

Experiments were conducted on mice, rats, rabbits, beagle dogs, rhesus monkeys, cockerels, and pullets, to determine the toxicity of chlordiazepoxide, alone and in combination with morphine, megimide, DL-amphetamine, caffeine, pentylenetetrazol, methyprylone, chloral hydrate, phenobarbital, glutethimide, and ethanol. For ethanol, the ip HD_{50} was determined, and the drug was then administered 10 min after 25 mg/kg chlordiazepoxide ip. The hypnotic effects of ethanol were only slightly potentiated. The effects of excessive doses of chlordiazepoxide in 22 suicide cases are also reported, including 10 cases of combinations of chlordiazepoxide and alcohol or another drug. The effects of these drug combinations are described.

1497. Zimmermann, E., and Remy, E.

UNTERSUCHUNG ÜBER DIE BEDEUTUNG DES ALKOHOLISMUS FÜR DIE ENTSTEHUNG EINER ARSENVERGIFTUNG. [Investigation of the significance of alcoholism for the development of arsenic poisoning].

Archiv für Gewerbepathologie und Gewerbehygiene (Berlin), 7: 486-496 (5 ref.), 1936.
G – exp. comp. – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mammals – chronic admin. – in vivo – blood lev. – other drug lev. – absorp., distrib., stor. – cardiovasc. – CNS – glands – liver, kidney – metab. proc. – skel., muscle, skin – unclass. ther. agents – *CAAAL-0 A-1465.

Over a period of 227-299 days, 4 pigs (average wt of 15 kg) of the same litter received a diet containing arsenic in the form of a 1% Fowler's sol. 2 of the animals also received beer (500 cc/day at first, and later 250 cc/day) in a single dose or in several smaller doses/day. The total amount of arsenic administered during the period was 150-350 g As_2O_3 . The animals showed no typical arsenic poisoning symptoms. Tolerance towards alcohol was greatly increased as the experiment progressed, but not, it was considered, as a result of the arsenic administration. When the animals were killed, neither dissection nor microscopic investigation revealed any significant findings. Chemical analysis for the presence of arsenic in the blood, muscle tissue, liver, spleen, kidneys, and brain, however, showed significantly higher concentrations in the alcohol-treated animals. It is concluded that alcohol induces greater retention of arsenic, through an unknown mechanism which disturbs arsenic elimination, and by increasing permeability of the intestinal wall, thereby enhancing arsenic absorption.

1498. Zipf, H. F., and Hamacher, J.

KOMBINATIONSEFFEKTE. I. MITTEILUNG: ALLGEMEINE FRAGEN DER KOMBINATIONSFORSCHUNG. [Combined effects. I. General questions on combinations research].

Arzneimittelforschung (Aulendorf), 15: 1267-1274 (22 ref.), 1965.

KOMBINATIONSEFFEKTE. 2. MITTEILUNG: EXPERIMENTELLE ERFASSUNG UND DARSTELLUNG VON KOMBINATIONSEFFEKTEN. [Combined effects. 2. Experimental recording and presentation of combined effects].

Arzneimittelforschung (Aulendorf), 16(3): 329-339 (56 ref.), 1966.

KOMBINATIONSEFFEKTE. 3. MITTEILUNG: SPEZIELLE FRAGEN DER KOMBINATIONSFORSCHUNG BEI ANTINEURALGISCHEN MISCHPRÄPARATEN, SONSTIGEN KOMBINATIONSPRÄPARATEN UND BEI NARKOSEKOMBINATIONEN. [Combined effects. 3. Special problems of research into combined effects of antineurologic combined drugs, other combined drugs, and combined drugs used in narcosis].

Arzneimittelforschung (Aulendorf), 16(10): 1297-1304 (40 ref.), 1966.

KOMBINATIONSEFFEKTE. 4. MITTEILUNG: VERKEHRSMEDIZINISCHE PROBLEME DES KOMBINATIONSEFFEKTES. [Combined effects. 4. Problem of synergism in traffic medicine].

Arzneimittelforschung (Aulendorf), 17(1): 70-79 (69 ref.), 1967.

G – review – cross-tol. – DC (decrease) – DC (supra-add. incr.) – DC (add., infra-add., unspec. incr.) – DC (unchanged) – mot. vehic. – humans – psychot. humans – mammals – blood lev. – mot. perform. – psychol. perform. – species or sex diff. – absorp., distrib., stor. – CNS – metab. proc. – analg., antipyret. – anesthetics – anticonvulsants – antidepressants – anti-infectants – antispasmodics –

autocoids – autonomic agents – barbiturates – cardiovasc. agents – gastrointest. agents – hormones, hormone antag. – indust. intox. – miscellaneous – musculoskel. agents – sed., hypnot. – stimulants – tranquilizers – unclass. ther. agents – *CAAAL-0 B-0493.

The first 3 communications discuss the problem of drug interaction in detail, and the fourth communication relates this problem to the question of drug combinations in traffic medicine. The fourth communication reviews the literature on the combined effects of a joint alcohol-drug intake or a joint intake of 2 or more drugs. The main topics reviewed are the interaction of alcohol and other drugs, atypical reactions caused by alcohol after prior drug intake, and the alteration of the blood alcohol content through drugs.

1499. Zirkle, G. A., King, P. D., McAtee, O. B., and Van Dyke, R.
EFFECTS OF CHLORPROMAZINE AND ALCOHOL ON COORDINATION AND JUDGMENT.
 J.A.M.A. (Chicago), 171: 1496-1499 (4 ref.), 1959.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – other drug lev. – mot. perform. – psychol. perform. – tranquilizers – *CAAAL-8887-J1 A-0411.

24 human subjects were exposed to the following experimental conditions: chlorpromazine placebo plus placebo drink, chlorpromazine (200 mg/day, begun 1 week prior to tests) plus placebo drink, chlorpromazine placebo plus alcohol drink (sufficient alcohol to produce a blood level of 0.05%), and chlorpromazine plus alcohol drink. The 4 conditions were rotated in 4 different sequences. Subjects were divided into 4 subgroups of 6, with each group assigned a different sequence. Placebos, chlorpromazine, and alcohol were administered po by a blind procedure. Neuromuscular coordination was then tested by 9 performance tests. The effect of alcohol and chlorpromazine, taken together, significantly impaired the performance of the subjects. The more complex tasks were the most affected, and the subjects became aware of dullness, lethargy, and poor coordination. Chlorpromazine alone impaired performance, but the impairment caused by alcohol with chlorpromazine was significantly greater. Patients obliged to take chlorpromazine should be warned that the use of alcohol greatly increases the dangers of operating complex machinery, including automobiles.

1500. Zirkle, G. A., McAtee, O. B., King, P. D., and Van Dyke, R.
MEPROBAMATE AND SMALL AMOUNTS OF ALCOHOL: EFFECTS ON HUMAN ABILITY, COORDINATION, AND JUDGMENT.
 J.A.M.A. (Chicago), 173: 1823-1825 (3 ref.), 1960.
 E – exp. cont. – DC (add., infra-add., unspec. incr.) – humans – acute admin. – in vivo – blood lev. – other drug lev. – psychol. perform. – tranquilizers – *CAAAL-9257-D1 A-1241.

22 normal human subjects were exposed to 4 experimental conditions: placebo drug plus placebo drink, meprobamate (400 mg, 4 times/day for 1 week before test) plus placebo drink, alcohol drink (sufficient alcohol to produce a concentration of 0.05% in the blood) plus placebo drug, and meprobamate plus alcohol. The 4 conditions were balanced by rotating them in 4 different sequences. The subjects were randomized into 4 subgroups of 5 or 6, with each subgroup assigned a different sequence. 8 psychological tests were administered blind. Performance, which was best in the placebo-placebo condition, deteriorated progressively under the meprobamate-placebo, alcohol-placebo, and alcohol-meprobamate conditions. Performance in the combined drug condition was significantly worse than in either of the separate drug conditions. Clinically, meprobamate and alcohol together produced more intoxication effects than alcohol alone in 16 subjects, 4 of whom appeared obviously drunk from the combination. Subjective effects correlated well with tests results and clinical observations. It is concluded that there is a supplementary, and possibly potentiating, effect of meprobamate on alcohol.

INDEXES

Key Word Index

QUALIFICATION OF PAPER

1. **SEC** secondary article (article in which the main aim is not to investigate or discuss the interaction of ethanol with other compounds, but some oblique reference to combined effects is made, or else the space pertinent to ethanol interaction is a relatively small part of the whole text)

1, 9, 19, 20, 21, 23, 24, 29, 32, 33, 63, 75, 79, 86, 97, 113, 115, 123, 124, 127, 128, 132, 162, 170, 172, 177, 190, 202, 209, 210, 212, 216, 255, 267, 268, 269, 271, 275, 283, 291, 296, 304, 307, 310, 321, 325, 333, 342, 348, 354, 360, 416, 418, 434, 436, 440, 445, 457, 458, 461, 464, 466, 498, 502, 503, 504, 518, 534, 539, 541, 546, 551, 562, 568, 570, 573, 576, 586, 588, 593, 595, 611, 619, 626, 632, 635, 636, 637, 638, 642, 649, 661, 662, 663, 680, 702, 725, 735, 740, 744, 747, 756, 770, 774, 794, 801, 802, 803, 817, 823, 835, 851, 861, 867, 874, 875, 885, 890, 892, 919, 920, 925, 928, 943, 952, 953, 955, 960, 970, 972, 985, 993, 1000, 1009, 1022, 1031, 1045, 1058, 1068, 1069, 1072, 1073, 1074, 1075, 1079, 1083, 1085, 1091, 1103, 1116, 1124, 1132, 1146, 1155, 1159, 1171, 1183, 1191, 1197, 1202, 1205, 1223, 1225, 1232, 1235, 1238, 1239, 1242, 1243, 1247, 1258, 1261, 1262, 1263, 1265, 1268, 1269, 1275, 1277, 1290, 1295, 1298, 1306, 1311, 1314, 1315, 1321, 1346, 1352, 1355, 1360, 1361, 1365, 1366, 1368, 1372, 1374, 1380, 1381, 1390, 1400, 1401, 1402, 1403, 1404, 1408, 1414, 1416, 1440, 1441, 1443, 1466, 1470, 1472, 1477, 1484

TYPE OF PAPER

2. **abst.** abstract only

27, 28, 148, 151, 166, 187, 190, 191, 194, 198, 218, 231, 294, 297, 397, 444, 467, 472, 473, 506, 513, 581, 630, 640, 651, 655, 658, 678, 703, 706, 716, 733, 777, 790, 812, 836, 838, 862, 894, 913, 914, 934, 935, 938, 948, 958, 961, 992, 1012, 1041, 1061, 1071, 1099, 1101, 1114, 1118, 1126, 1130, 1157, 1159, 1161, 1167, 1171, 1190, 1216, 1217, 1247, 1253, 1282, 1285, 1320, 1330, 1332, 1336, 1337, 1359, 1374, 1376, 1394, 1396, 1409, 1410, 1417, 1424, 1433, 1436, 1439, 1456, 1457, 1465, 1469, 1488, 1495

3. **exp. cont.** experimental study, controlled
4, 8, 14, 15, 16, 17, 18, 27, 29, 34, 35, 61, 76, 77, 80, 81, 84, 85, 87, 88, 89, 90, 92, 93, 94, 99, 100, 101, 108, 109, 110, 111, 113, 114, 116, 117, 118, 119, 122, 126, 130, 131, 136, 137, 138, 142, 143, 144, 147, 156, 159, 160, 163, 164, 168, 171, 174, 176, 177, 178, 180, 184, 186, 187, 188, 189, 192, 197, 200, 201, 205, 206, 211, 212, 213, 215, 220, 225, 227, 231, 235, 237, 242, 247, 254, 257, 258, 261, 262, 263, 264, 265, 266, 272, 273, 275, 279, 280, 281, 287, 288, 289, 291, 292, 293, 294, 295, 297, 299, 303, 304, 311, 313, 316, 317, 318, 319, 320, 323, 326, 327, 331, 335, 337, 338, 339, 340, 341, 344, 356, 360, 361, 364, 365, 370, 371, 372, 373, 377, 383, 388, 389, 390, 394, 396, 400, 401, 406, 407, 408, 409, 415, 417, 419, 420, 423, 424, 425, 427, 428, 433, 438, 441, 443, 444, 448, 449, 450, 451, 452, 453, 454, 459, 463, 464, 465, 467, 468, 469, 473, 476, 479, 480, 481, 485, 486, 487, 488, 489, 490, 491, 492, 493, 494, 495, 496, 497, 501, 504, 505, 506, 508, 513, 514, 515, 519, 522, 526, 528, 529, 535, 537, 538, 540, 541, 543, 544, 547, 548, 549, 559, 560, 561, 564, 565, 566, 567, 569, 573, 576, 579, 580, 581, 583, 584, 585, 587, 588, 596, 597, 598, 599, 601, 602, 603, 604, 605, 608, 609, 610, 612, 615, 616, 617, 622, 624, 629, 633, 634, 637, 640, 641, 643, 644, 645, 646, 647, 648, 650, 651, 652, 653, 655, 656, 657, 658, 659, 661, 662, 663, 664, 666, 671, 672, 676, 677, 679, 681, 682, 684, 685, 686, 687, 689, 692, 694, 698, 699, 700, 702, 704, 706, 707, 710, 714, 715, 716, 718, 720, 726, 727, 730, 736, 738, 740, 741, 742, 745, 746, 749, 750, 754, 755, 756, 760, 766, 769, 771, 774, 778, 779, 780, 787, 788, 789, 792, 797, 799, 804, 811, 812, 815, 816, 818, 819, 822, 823, 825, 826, 827, 831, 833, 835, 837, 838, 840, 841, 843, 847, 848, 854, 858, 863, 867, 870, 873, 874, 875, 876, 877, 878, 879, 881, 882, 883, 886, 888, 889, 890, 892, 894, 895, 896, 898, 899, 900, 901, 902, 903, 905, 919, 921, 923, 927, 931, 932, 936, 937, 939, 940, 944, 945, 946, 947, 957, 962, 963, 969, 973, 974, 978, 979, 980, 981, 982, 984, 988, 990, 991, 992, 995, 998, 999, 1015, 1016, 1017, 1018, 1020, 1021, 1022, 1024, 1027, 1033, 1036, 1039, 1040, 1042, 1043, 1044, 1046, 1047, 1051, 1053, 1054, 1057, 1059, 1062, 1064, 1065, 1073,

- 1074, 1077, 1078, 1084, 1086, 1087, 1089, 1090, 1092, 1093, 1097, 1098, 1101, 1102, 1103, 1104, 1106, 1107, 1110, 1112, 1113, 1114, 1117, 1123, 1126, 1127, 1128, 1131, 1145, 1147, 1149, 1150, 1151, 1152, 1153, 1156, 1158, 1159, 1161, 1162, 1163, 1165, 1166, 1167, 1168, 1170, 1172, 1174, 1175, 1177, 1178, 1180, 1182, 1185, 1188, 1189, 1191, 1192, 1194, 1195, 1196, 1200, 1206, 1210, 1211, 1213, 1214, 1218, 1220, 1224, 1227, 1228, 1231, 1232, 1233, 1237, 1245, 1247, 1255, 1256, 1257, 1260, 1264, 1266, 1267, 1270, 1271, 1272, 1273, 1286, 1287, 1290, 1291, 1292, 1294, 1296, 1301, 1302, 1304, 1305, 1307, 1308, 1310, 1312, 1316, 1317, 1318, 1319, 1320, 1321, 1325, 1326, 1327, 1328, 1329, 1330, 1333, 1334, 1337, 1340, 1341, 1343, 1344, 1349, 1350, 1354, 1356, 1362, 1363, 1364, 1367, 1369, 1373, 1374, 1379, 1381, 1382, 1383, 1384, 1386, 1388, 1389, 1390, 1391, 1392, 1393, 1396, 1398, 1405, 1407, 1411, 1412, 1415, 1416, 1417, 1418, 1419, 1420, 1421, 1422, 1424, 1425, 1426, 1427, 1428, 1432, 1437, 1438, 1442, 1444, 1445, 1446, 1447, 1449, 1451, 1452, 1453, 1454, 1455, 1462, 1463, 1472, 1474, 1475, 1476, 1480, 1482, 1486, 1487, 1490, 1491, 1493, 1494, 1496, 1499, 1500
4. **exp. comp.** experimental study, comparative
2, 5, 7, 8, 10, 15, 25, 28, 31, 36, 54, 61, 76, 79, 85, 87, 89, 90, 92, 94, 98, 101, 102, 110, 113, 114, 118, 129, 136, 137, 140, 150, 158, 160, 161, 163, 167, 173, 177, 181, 184, 187, 188, 194, 198, 201, 203, 221, 222, 224, 235, 238, 239, 241, 242, 243, 244, 246, 247, 248, 254, 257, 258, 261, 262, 264, 265, 266, 272, 273, 276, 278, 279, 280, 286, 287, 288, 289, 292, 297, 303, 305, 311, 313, 316, 317, 318, 319, 322, 323, 328, 329, 334, 337, 344, 355, 356, 357, 364, 365, 366, 369, 371, 380, 381, 382, 383, 384, 387, 391, 396, 397, 399, 401, 405, 406, 407, 414, 417, 421, 423, 425, 438, 441, 451, 452, 453, 455, 468, 469, 472, 474, 475, 477, 478, 489, 492, 493, 494, 495, 496, 501, 504, 506, 508, 510, 512, 513, 514, 515, 524, 530, 537, 539, 540, 542, 547, 557, 559, 561, 564, 567, 569, 572, 574, 575, 576, 577, 579, 584, 597, 598, 600, 602, 604, 605, 607, 610, 611, 612, 613, 614, 617, 621, 623, 625, 629, 631, 635, 640, 644, 656, 659, 661, 676, 681, 682, 684, 685, 686, 687, 689, 694, 697, 699, 701, 709, 710, 714, 715, 721, 722, 730, 737, 742, 746, 749, 751, 757, 760, 761, 762, 763, 764, 765, 766, 768, 769, 776, 777, 778, 780, 782, 787, 789, 792, 798, 804, 806, 812, 816, 819, 824, 825, 826, 827, 836, 837, 845, 847, 848, 858, 863, 867, 871, 872, 874, 875, 878, 882, 883, 891, 904, 921, 933, 935, 936, 937, 939, 940, 941, 942, 945, 946, 948, 955, 957, 967, 969, 973, 978, 979, 984, 989, 991, 993, 996, 1011, 1012, 1014, 1016, 1017, 1020, 1022, 1030, 1035, 1041, 1042, 1046, 1047, 1054, 1057, 1068, 1073, 1077, 1078, 1084, 1087, 1089, 1090, 1091, 1092, 1095, 1097, 1101, 1109, 1114, 1116, 1125, 1126, 1127, 1128, 1130, 1131, 1149, 1156, 1157, 1163, 1174, 1175, 1176, 1177, 1178, 1179, 1181, 1182, 1183, 1188, 1189, 1190, 1192, 1201, 1204, 1206, 1208, 1209, 1211, 1212, 1216, 1217, 1221, 1225, 1229, 1230, 1231, 1234, 1240, 1241, 1243, 1246, 1247, 1248, 1252, 1266, 1267, 1268, 1271, 1273, 1286, 1287, 1299, 1300, 1301, 1307, 1308, 1315, 1324, 1325, 1327, 1332, 1339, 1343, 1349, 1356, 1359, 1363, 1374, 1375, 1376, 1379, 1386, 1388, 1390, 1391, 1394, 1397, 1398, 1405, 1409, 1425, 1426, 1427, 1428, 1433, 1437, 1443, 1444, 1446, 1449, 1451, 1452, 1456, 1457, 1461, 1463, 1466, 1467, 1475, 1479, 1486, 1488, 1493, 1496, 1497
5. **exp.** experimental study, other than items 3 and 4
19, 22, 59, 78, 82, 86, 103, 106, 128, 135, 141, 148, 149, 151, 183, 190, 191, 207, 217, 218, 219, 230, 232, 233, 234, 236, 253, 300, 306, 324, 378, 386, 395, 422, 434, 466, 503, 509, 517, 518, 533, 546, 556, 562, 568, 578, 582, 591, 618, 627, 660, 665, 678, 703, 713, 717, 723, 724, 732, 747, 748, 752, 772, 800, 805, 820, 828, 829, 830, 862, 868, 893, 911, 930, 950, 951, 954, 961, 964, 971, 975, 976, 997, 1010, 1019, 1025, 1026, 1028, 1055, 1083, 1099, 1105, 1121, 1122, 1132, 1148, 1160, 1164, 1173, 1186, 1187, 1198, 1199, 1207, 1222, 1244, 1249, 1250, 1254, 1306, 1331, 1336, 1342, 1358, 1399, 1410, 1413, 1431, 1436, 1439, 1450, 1458, 1459, 1460, 1464, 1465, 1468, 1478, 1481, 1495
6. **general** general paper, no experimental or statistical research
1, 3, 6, 9, 11, 12, 13, 26, 30, 32, 33, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 55, 56, 57, 58, 59, 60, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 83, 91, 95, 96, 104, 105, 107, 112, 115, 120, 123, 124, 127, 132, 133, 134, 139, 140, 145, 146, 149, 152, 153, 154, 156, 157, 165, 169, 170, 172, 185, 193, 196, 197, 199, 204, 208, 209, 210,

- 223, 226, 228, 229, 240, 245, 250, 251, 252, 256, 259, 260, 268, 271, 274, 277, 282, 283, 284, 285, 290, 296, 301, 302, 307, 308, 309, 312, 315, 325, 332, 333, 336, 342, 345, 346, 347, 349, 350, 351, 353, 362, 363, 374, 376, 379, 385, 392, 393, 398, 402, 410, 412, 413, 416, 418, 426, 429, 430, 432, 435, 436, 437, 439, 440, 442, 447, 456, 458, 460, 461, 462, 470, 471, 482, 483, 484, 507, 516, 523, 525, 527, 530, 531, 532, 536, 539, 545, 550, 551, 552, 553, 554, 555, 558, 563, 568, 570, 578, 586, 589, 590, 591, 592, 593, 594, 595, 606, 620, 628, 630, 632, 636, 638, 639, 654, 665, 667, 668, 669, 670, 675, 683, 688, 690, 691, 695, 696, 705, 708, 711, 712, 717, 718, 724, 725, 728, 729, 731, 732, 733, 734, 735, 739, 743, 744, 753, 758, 770, 772, 773, 781, 783, 784, 785, 791, 793, 794, 795, 796, 800, 802, 803, 807, 808, 809, 810, 813, 814, 821, 832, 834, 842, 844, 846, 849, 850, 852, 853, 855, 856, 857, 859, 860, 861, 864, 866, 869, 878, 880, 885, 887, 895, 897, 907, 908, 909, 910, 912, 917, 918, 920, 922, 924, 925, 926, 949, 952, 953, 956, 958, 959, 964, 965, 966, 968, 970, 971, 972, 977, 985, 986, 994, 1000, 1001, 1002, 1003, 1004, 1005, 1006, 1007, 1008, 1009, 1013, 1023, 1025, 1026, 1028, 1029, 1031, 1032, 1034, 1037, 1038, 1045, 1048, 1049, 1050, 1056, 1060, 1061, 1066, 1067, 1070, 1071, 1072, 1076, 1079, 1088, 1094, 1100, 1115, 1118, 1119, 1120, 1124, 1129, 1133, 1134, 1135, 1136, 1140, 1141, 1142, 1143, 1144, 1146, 1154, 1155, 1156, 1169, 1171, 1185, 1193, 1197, 1202, 1203, 1205, 1219, 1226, 1235, 1236, 1238, 1239, 1242, 1243, 1248, 1263, 1265, 1269, 1274, 1275, 1276, 1278, 1279, 1280, 1281, 1282, 1283, 1284, 1285, 1288, 1289, 1293, 1295, 1303, 1309, 1311, 1314, 1335, 1338, 1345, 1347, 1348, 1351, 1352, 1353, 1357, 1360, 1361, 1366, 1368, 1370, 1371, 1372, 1377, 1378, 1380, 1385, 1387, 1395, 1399, 1400, 1402, 1404, 1406, 1408, 1414, 1434, 1435, 1440, 1441, 1470, 1471, 1473, 1477, 1483, 1484, 1485, 1489, 1492
 7. **presentation** presentation, unpublished or not published in a regular journal, made before a conference, a seminar, or a session of an organization
130, 214, 261, 298, 337, 422, 429, 430, 445, 493, 494, 495, 511, 600, 650, 659, 685, 686, 732, 733, 775, 804, 831, 840, 939, 987, 1116, 1215, 1322, 1362, 1394, 1396, 1406, 1433, 1441, 1469
 8. **review** review of literature
54, 61, 121, 125, 179, 182, 195, 196, 214, 216, 223, 226, 229, 267, 269, 330, 343, 347, 348, 351, 352, 354, 367, 368, 375, 418, 426, 431, 432, 446, 457, 498, 499, 500, 502, 504, 511, 520, 521, 526, 553, 570, 582, 591, 592, 595, 626, 674, 680, 683, 693, 694, 719, 775, 783, 784, 785, 786, 790, 801, 807, 839, 864, 865, 906, 928, 929, 954, 960, 983, 985, 1037, 1052, 1063, 1068, 1080, 1081, 1082, 1085, 1096, 1113, 1142, 1164, 1169, 1277, 1313, 1358, 1380, 1423, 1429, 1448, 1498
 9. **stat. surv.** survey or statistical research
91, 97, 155, 175, 249, 255, 270, 298, 314, 321, 358, 359, 403, 404, 411, 531, 534, 571, 619, 642, 673, 759, 767, 829, 851, 869, 884, 914, 915, 916, 987, 1069, 1085, 1108, 1111, 1137, 1138, 1139, 1184, 1223, 1251, 1259, 1261, 1262, 1297, 1298, 1322, 1323, 1346, 1401, 1403, 1430
- SUBJECT OF PAPER
10. **case hist.** case histories (concerning treatment of intoxication or toxic effects)
1, 6, 11, 12, 13, 23, 24, 26, 37, 38, 63, 70, 96, 120, 124, 127, 140, 153, 157, 185, 193, 224, 227, 228, 245, 250, 251, 256, 274, 277, 283, 285, 342, 363, 379, 410, 412, 413, 435, 437, 440, 442, 456, 461, 462, 466, 470, 471, 483, 484, 516, 518, 523, 525, 530, 531, 532, 545, 550, 551, 589, 590, 594, 636, 649, 654, 670, 696, 726, 728, 793, 795, 796, 821, 842, 844, 846, 849, 850, 853, 856, 857, 859, 860, 861, 864, 897, 917, 918, 922, 949, 953, 959, 968, 1009, 1013, 1023, 1031, 1038, 1048, 1050, 1060, 1061, 1072, 1094, 1119, 1120, 1135, 1136, 1137, 1138, 1141, 1142, 1158, 1171, 1193, 1205, 1219, 1238, 1242, 1263, 1276, 1306, 1368, 1370, 1378, 1385, 1387, 1395, 1414, 1471, 1484, 1485
 11. **congen. stud.** congener studies
2, 3, 79, 88, 98, 110, 141, 150, 183, 184, 212, 230, 244, 287, 288, 289, 290, 317, 318, 331, 380, 381, 382, 383, 391, 400, 478, 492, 493, 505, 510, 511, 512, 537, 541, 542, 582, 610, 659, 660, 687, 697, 698, 699, 700, 767, 778, 919, 934, 935, 936, 937, 938, 939, 940, 941, 946, 951, 954, 967, 978, 1012, 1097, 1175, 1176, 1177, 1192, 1200, 1234, 1243, 1260, 1339, 1388, 1389, 1423, 1443, 1461, 1474
 12. **conj. addict.** conjunctive addiction (addiction to alcohol plus other drugs)
32, 38, 63, 97, 123, 179, 249, 282, 285, 308,

314, 315, 321, 347, 351, 398, 402, 403, 517,
545, 636, 668, 669, 849, 850, 916, 1009, 1050,
1146, 1223, 1261, 1262, 1345, 1351, 1372,
1471

13. **cross-tol.** cross-tolerance

9, 14, 15, 80, 135, 162, 166, 168, 231, 315,
443, 445, 561, 570, 586, 617, 637, 646, 655,
656, 657, 658, 677, 719, 766, 774, 787, 802,
803, 852, 861, 864, 894, 955, 956, 983, 1037,
1093, 1102, 1117, 1129, 1130, 1154, 1161,
1162, 1163, 1164, 1165, 1167, 1237, 1266,
1268, 1306, 1311, 1330, 1350, 1382, 1392,
1409, 1412, 1423, 1469, 1479, 1498

14. **DC (antidotal)** drug combinations—antidotal
use in clinical treatment (exclude item 15)

6, 11, 12, 13, 30, 70, 95, 125, 139, 153, 154,
163, 169, 172, 193, 217, 218, 219, 223, 224,
226, 227, 228, 229, 236, 250, 251, 256, 260,
269, 284, 307, 325, 334, 352, 353, 379, 412,
431, 442, 461, 462, 471, 482, 483, 484, 523,
545, 550, 582, 589, 590, 594, 620, 649, 670,
696, 701, 717, 718, 719, 726, 728, 732, 733,
773, 791, 793, 809, 844, 853, 859, 860, 871,
872, 876, 906, 917, 918, 922, 949, 966, 968,
986, 994, 1000, 1013, 1023, 1037, 1038, 1045,
1058, 1060, 1061, 1063, 1072, 1094, 1096,
1105, 1106, 1120, 1123, 1124, 1134, 1135,
1136, 1137, 1138, 1139, 1140, 1141, 1142,
1143, 1144, 1222, 1242, 1276, 1288, 1292,
1303, 1319, 1345, 1347, 1348, 1370, 1377,
1378, 1387, 1395, 1406, 1415, 1423, 1484,
1485

15. **DC (decrease)** drug combinations—decrease
of drug effects or of toxic effect on organism
(exclude item 14)

4, 10, 22, 25, 31, 53, 54, 58, 59, 61, 62, 68,
72, 73, 76, 78, 81, 82, 84, 85, 87, 89, 90, 92,
100, 101, 102, 103, 111, 113, 116, 117, 118,
121, 122, 130, 133, 136, 138, 140, 143, 173,
178, 181, 182, 194, 195, 205, 211, 213, 216,
217, 218, 219, 220, 221, 222, 223, 224, 225,
226, 227, 229, 232, 233, 234, 235, 236, 237,
243, 247, 248, 257, 258, 266, 274, 276, 278,
280, 281, 294, 303, 305, 308, 309, 334, 335,
337, 338, 339, 340, 361, 364, 366, 367, 368,
369, 370, 372, 373, 374, 375, 389, 390, 401,
405, 415, 418, 422, 423, 426, 431, 432, 434,
438, 441, 446, 450, 452, 453, 454, 458, 463,
475, 477, 480, 481, 485, 486, 487, 488, 489,
490, 494, 495, 496, 499, 500, 504, 506, 513,
514, 522, 535, 536, 538, 539, 540, 543, 547,
548, 549, 556, 557, 560, 561, 562, 564, 569,
572, 574, 577, 582, 591, 595, 602, 605, 609,
614, 615, 616, 618, 622, 625, 626, 627, 628,

629, 635, 650, 671, 672, 674, 690, 701, 707,
710, 713, 717, 718, 719, 721, 722, 723, 724,
730, 734, 737, 746, 755, 760, 761, 762, 763,
764, 765, 769, 771, 775, 782, 790, 792, 807,
808, 812, 819, 826, 827, 830, 831, 833, 838,
839, 841, 852, 858, 864, 865, 868, 882, 889,
891, 893, 894, 913, 924, 925, 932, 945, 946,
950, 951, 957, 960, 964, 969, 971, 973, 974,
976, 979, 980, 981, 983, 985, 988, 989, 993,
996, 998, 1019, 1030, 1034, 1036, 1037, 1041,
1053, 1054, 1055, 1059, 1063, 1069, 1074,
1078, 1083, 1084, 1092, 1095, 1100, 1107,
1110, 1122, 1123, 1125, 1126, 1127, 1128,
1131, 1138, 1139, 1142, 1143, 1144, 1145,
1148, 1149, 1151, 1152, 1170, 1172, 1173,
1174, 1182, 1190, 1194, 1195, 1196, 1204,
1206, 1209, 1211, 1220, 1224, 1232, 1233,
1240, 1241, 1242, 1244, 1246, 1253, 1255,
1256, 1257, 1264, 1267, 1269, 1271, 1272,
1279, 1280, 1281, 1283, 1284, 1287, 1294,
1295, 1296, 1304, 1316, 1317, 1318, 1320,
1321, 1331, 1336, 1337, 1340, 1341, 1351,
1358, 1359, 1363, 1367, 1373, 1384, 1386,
1391, 1393, 1394, 1406, 1411, 1413, 1417,
1419, 1420, 1423, 1425, 1426, 1437, 1439,
1444, 1450, 1451, 1452, 1456, 1457, 1466,
1467, 1470, 1476, 1479, 1480, 1481, 1486,
1487, 1488, 1489, 1491, 1493, 1494, 1495,
1498

16. **DC (supra-add. incr.)** drug

combinations—supra-additive (more than
additive) increase of drug effects or of toxic
effect on organism (if specifically stated as
such in paper)

66, 81, 93, 102, 126, 133, 316, 330, 343, 346,
355, 365, 431, 468, 475, 488, 500, 566, 571,
582, 612, 653, 709, 715, 719, 832, 839, 948,
985, 1014, 1015, 1016, 1064, 1153, 1188, 1211,
1218, 1270, 1274, 1315, 1359, 1380, 1406,
1407, 1423, 1424, 1441, 1463, 1464, 1498

17. **DC (add., infra-add., unspec. incr.)** drug
combinations—additive, infra-additive (less
than additive), or unspecified increase of drug
effects or of toxic effect on organism

1, 5, 16, 18, 20, 21, 26, 28, 29, 32, 33, 34, 35,
36, 37, 38, 39, 40, 41, 43, 44, 45, 46, 47, 48,
49, 50, 51, 52, 53, 55, 56, 57, 58, 62, 63, 66,
67, 68, 72, 75, 77, 78, 82, 83, 87, 91, 92, 93,
94, 96, 98, 99, 105, 115, 118, 119, 120, 121,
127, 129, 133, 134, 137, 142, 147, 148, 151,
155, 157, 158, 159, 160, 165, 167, 175, 176,
177, 179, 181, 185, 187, 188, 189, 190, 191,
192, 195, 196, 197, 203, 205, 206, 214, 215,
216, 221, 222, 234, 239, 242, 243, 245, 247,

249, 254, 257, 261, 262, 263, 264, 265, 267,
268, 270, 271, 272, 273, 275, 276, 279, 289,
292, 293, 295, 297, 299, 300, 310, 311, 312,
319, 323, 326, 327, 328, 329, 330, 333, 336,
337, 338, 339, 340, 343, 344, 345, 346, 347,
348, 349, 350, 351, 354, 356, 357, 358, 359,
360, 361, 362, 363, 364, 365, 366, 367, 368,
372, 375, 376, 377, 378, 385, 386, 388, 389,
392, 395, 396, 399, 403, 405, 411, 416, 421,
422, 423, 424, 425, 426, 427, 429, 430, 431,
432, 436, 437, 440, 441, 444, 446, 447, 448,
452, 455, 456, 459, 460, 464, 465, 467, 468,
469, 470, 472, 473, 474, 475, 476, 479, 485,
486, 487, 491, 492, 493, 494, 495, 496, 497,
498, 500, 501, 502, 505, 506, 507, 508, 518,
519, 520, 521, 522, 523, 524, 526, 527, 528,
529, 531, 532, 534, 535, 540, 547, 551, 552,
553, 554, 558, 564, 565, 567, 569, 570, 572,
573, 574, 575, 576, 578, 579, 580, 582, 583,
584, 587, 588, 591, 592, 593, 595, 597, 598,
600, 603, 604, 605, 611, 612, 619, 620, 621,
623, 625, 626, 628, 631, 632, 633, 634, 635,
641, 642, 644, 645, 647, 648, 651, 652, 653,
654, 664, 665, 666, 667, 673, 674, 676, 678,
679, 680, 683, 684, 693, 694, 698, 700, 706,
712, 714, 715, 716, 717, 718, 719, 720, 721,
722, 725, 726, 731, 736, 738, 739, 740, 741,
742, 743, 745, 747, 749, 750, 756, 758, 759,
760, 761, 763, 764, 766, 768, 769, 770, 775,
776, 777, 783, 784, 785, 790, 792, 794, 797,
800, 801, 804, 805, 806, 807, 811, 813, 814,
816, 817, 818, 820, 821, 822, 823, 824, 826,
827, 829, 832, 833, 834, 835, 837, 839, 842,
843, 846, 847, 848, 854, 855, 857, 858, 861,
862, 863, 869, 870, 877, 878, 879, 880, 881,
882, 883, 884, 885, 886, 887, 888, 890, 891,
892, 897, 898, 899, 900, 902, 903, 904, 905,
907, 908, 909, 910, 912, 913, 914, 915, 920,
921, 923, 924, 925, 926, 928, 929, 930, 931,
932, 933, 943, 945, 946, 947, 952, 953, 957,
958, 959, 961, 962, 963, 965, 966, 970, 971,
972, 975, 977, 982, 983, 985, 987, 989, 991,
992, 997, 999, 1001, 1002, 1003, 1004, 1005,
1006, 1007, 1008, 1011, 1016, 1017, 1018,
1020, 1021, 1022, 1024, 1025, 1026, 1027,
1028, 1029, 1031, 1032, 1033, 1034, 1035,
1037, 1041, 1044, 1046, 1047, 1048, 1049,
1050, 1051, 1052, 1056, 1057, 1059, 1062,
1063, 1065, 1066, 1067, 1068, 1070, 1071,
1073, 1074, 1075, 1076, 1077, 1078, 1079,
1085, 1086, 1087, 1088, 1089, 1090, 1092,
1098, 1099, 1100, 1103, 1108, 1111, 1112,
1113, 1115, 1117, 1118, 1121, 1128, 1132,
1133, 1142, 1143, 1144, 1147, 1149, 1150,

1155, 1160, 1164, 1166, 1168, 1169, 1170,
1178, 1180, 1181, 1182, 1183, 1184, 1185,
1186, 1189, 1190, 1197, 1198, 1199, 1201,
1202, 1205, 1208, 1209, 1210, 1211, 1213,
1214, 1215, 1216, 1217, 1219, 1221, 1223,
1225, 1226, 1227, 1231, 1232, 1236, 1239,
1240, 1241, 1251, 1252, 1253, 1254, 1255,
1256, 1257, 1258, 1259, 1260, 1263, 1265,
1267, 1269, 1273, 1274, 1275, 1277, 1278,
1279, 1280, 1281, 1282, 1283, 1284, 1285,
1286, 1289, 1290, 1291, 1293, 1296, 1297,
1301, 1302, 1305, 1307, 1308, 1309, 1310,
1311, 1312, 1313, 1314, 1315, 1320, 1321,
1322, 1323, 1325, 1326, 1327, 1328, 1332,
1333, 1334, 1335, 1338, 1342, 1343, 1344,
1349, 1352, 1353, 1354, 1356, 1357, 1358,
1360, 1361, 1362, 1368, 1369, 1371, 1374,
1375, 1376, 1380, 1383, 1384, 1390, 1391,
1396, 1399, 1400, 1401, 1402, 1403, 1404,
1406, 1407, 1408, 1410, 1412, 1417, 1419,
1422, 1423, 1428, 1429, 1430, 1431, 1432,
1433, 1434, 1435, 1436, 1437, 1438, 1440,
1441, 1442, 1443, 1446, 1447, 1448, 1449,
1452, 1454, 1459, 1460, 1462, 1464, 1465,
1472, 1473, 1475, 1477, 1479, 1480, 1482,
1483, 1491, 1492, 1493, 1496, 1497, 1498,
1499, 1500

18. DC (unchanged) drug

combinations—unchanged drug effects

7, 8, 17, 19, 22, 25, 36, 58, 59, 62, 64, 65, 71,
72, 81, 82, 87, 100, 131, 144, 149, 152, 156,
158, 160, 161, 164, 167, 170, 171, 173, 174,
177, 180, 186, 188, 194, 195, 202, 206, 214,
216, 234, 235, 238, 240, 241, 242, 243, 246,
247, 250, 253, 254, 265, 270, 272, 273, 276,
291, 306, 313, 334, 338, 339, 340, 341, 345,
368, 369, 371, 373, 374, 375, 384, 386, 394,
396, 399, 413, 417, 419, 420, 421, 422, 423,
426, 428, 432, 435, 439, 449, 451, 452, 453,
457, 459, 500, 503, 509, 515, 516, 533, 536,
556, 557, 559, 561, 574, 582, 585, 587, 591,
596, 599, 600, 601, 603, 604, 605, 607, 608,
613, 614, 618, 623, 629, 630, 644, 647, 661,
662, 663, 664, 674, 676, 677, 681, 682, 692,
701, 702, 717, 723, 724, 726, 727, 734, 746,
752, 754, 757, 761, 762, 765, 766, 769, 772,
775, 776, 779, 780, 792, 797, 799, 804, 805,
807, 815, 816, 819, 825, 826, 827, 828, 830,
838, 839, 840, 841, 843, 845, 865, 867, 873,
874, 875, 882, 895, 896, 901, 924, 927, 928,
942, 944, 962, 963, 964, 973, 979, 983, 984,
990, 991, 995, 1004, 1010, 1021, 1039, 1040,
1042, 1043, 1065, 1073, 1077, 1084, 1089,
1092, 1099, 1104, 1107, 1110, 1116, 1151,

- 1152, 1163, 1173, 1191, 1194, 1197, 1204, 1206, 1211, 1212, 1216, 1217, 1228, 1229, 1230, 1231, 1245, 1247, 1267, 1271, 1272, 1273, 1279, 1283, 1304, 1320, 1325, 1328, 1330, 1331, 1336, 1349, 1355, 1361, 1366, 1367, 1375, 1391, 1405, 1413, 1415, 1417, 1418, 1419, 1420, 1421, 1425, 1426, 1427, 1428, 1430, 1432, 1444, 1445, 1452, 1453, 1455, 1458, 1467, 1478, 1481, 1490, 1491, 1492, 1494, 1497, 1498
19. **DC (unspec.)** drug
combinations—unspecified drug effects
23, 24, 27, 74, 112, 121, 204, 248, 283, 296, 298, 301, 302, 304, 320, 322, 324, 342, 387, 393, 404, 406, 407, 408, 409, 517, 527, 640, 675, 680, 688, 691, 708, 735, 748, 836, 856, 1109, 1114, 1171, 1187, 1203, 1207, 1298, 1365, 1381, 1389, 1414
20. **DC (sensit.)** drug
combinations—sensitization or intolerance to alcohol
16, 17, 42, 60, 69, 104, 105, 106, 107, 108, 109, 113, 114, 128, 132, 133, 145, 146, 171, 195, 198, 199, 200, 201, 207, 208, 209, 210, 248, 252, 255, 259, 262, 277, 284, 286, 319, 332, 334, 341, 397, 410, 414, 431, 433, 446, 466, 469, 500, 520, 521, 525, 543, 544, 555, 563, 568, 575, 582, 606, 624, 625, 626, 638, 639, 643, 683, 689, 693, 695, 703, 704, 705, 711, 719, 720, 725, 726, 729, 744, 751, 753, 781, 783, 784, 785, 795, 796, 798, 810, 861, 866, 911, 965, 1034, 1037, 1063, 1080, 1081, 1082, 1100, 1118, 1119, 1156, 1157, 1158, 1193, 1235, 1238, 1248, 1249, 1250, 1259, 1283, 1299, 1300, 1324, 1346, 1364, 1379, 1385, 1397, 1398, 1406, 1423, 1434, 1435, 1468
21. **med.-leg.** medico-legal
23, 24, 43, 46, 51, 53, 55, 56, 57, 62, 83, 142, 175, 196, 239, 321, 329, 333, 336, 346, 348, 358, 362, 403, 404, 431, 436, 460, 520, 521, 531, 552, 553, 554, 563, 578, 591, 680, 713, 724, 735, 758, 801, 807, 869, 870, 907, 908, 909, 924, 925, 947, 985, 987, 1001, 1002, 1003, 1004, 1005, 1006, 1007, 1008, 1024, 1025, 1026, 1028, 1049, 1051, 1066, 1067, 1073, 1076, 1111, 1184, 1197, 1201, 1202, 1203, 1223, 1258, 1279, 1283, 1408, 1429, 1430, 1455
22. **mot. vehic.** operation of motor vehicles or simulators—including all forms of motor transport
43, 44, 48, 51, 53, 55, 56, 57, 62, 68, 131, 180, 214, 298, 315, 326, 327, 333, 336, 343, 344, 348, 362, 363, 370, 403, 404, 426, 431, 494, 520, 521, 531, 552, 553, 554, 591, 592, 593, 596, 604, 605, 619, 628, 629, 673, 680, 681, 682, 683, 684, 685, 686, 694, 724, 745, 801, 804, 807, 834, 843, 869, 870, 874, 875, 880, 884, 888, 924, 925, 928, 929, 970, 972, 985, 987, 1001, 1002, 1003, 1004, 1005, 1006, 1007, 1008, 1024, 1025, 1026, 1028, 1039, 1066, 1067, 1068, 1070, 1108, 1111, 1169, 1174, 1176, 1203, 1258, 1261, 1262, 1269, 1279, 1282, 1283, 1297, 1298, 1322, 1323, 1333, 1334, 1400, 1401, 1403, 1404, 1406, 1408, 1429, 1430, 1498
23. **post-mort.** post-mortem findings in
interaction poisonings
1, 26, 120, 124, 127, 155, 157, 163, 172, 175, 228, 251, 270, 298, 346, 379, 437, 507, 563, 571, 632, 636, 649, 712, 758, 814, 821, 842, 846, 907, 908, 909, 912, 915, 920, 943, 1137, 1138, 1147, 1184, 1205, 1259, 1353, 1368

SUBJECTS OR BEINGS AFFECTED BY INTERACTION

24. **humans** humans, healthy or to whom items 25 and 26 do not apply
1, 2, 3, 6, 13, 22, 23, 24, 30, 32, 33, 36, 37, 39, 40, 41, 42, 43, 44, 45, 47, 48, 49, 50, 51, 52, 53, 56, 57, 58, 59, 60, 61, 62, 64, 65, 66, 67, 68, 69, 72, 73, 74, 83, 85, 87, 91, 95, 96, 99, 100, 104, 105, 106, 107, 108, 110, 112, 114, 115, 117, 120, 121, 124, 125, 128, 129, 130, 131, 132, 133, 134, 136, 137, 141, 142, 144, 145, 146, 147, 148, 154, 155, 156, 157, 158, 161, 163, 164, 165, 169, 171, 172, 174, 175, 178, 180, 182, 183, 184, 185, 186, 188, 189, 190, 192, 193, 195, 198, 199, 200, 202, 206, 207, 208, 209, 210, 212, 213, 214, 217, 218, 219, 223, 224, 226, 227, 228, 229, 231, 235, 236, 237, 238, 239, 240, 244, 246, 250, 251, 252, 253, 255, 256, 259, 260, 268, 269, 270, 271, 276, 277, 283, 286, 288, 289, 290, 293, 296, 301, 302, 306, 309, 310, 311, 312, 319, 324, 326, 327, 328, 329, 330, 331, 332, 333, 334, 335, 336, 337, 338, 339, 340, 342, 343, 344, 345, 346, 348, 349, 350, 352, 353, 354, 358, 359, 361, 362, 367, 368, 369, 370, 371, 372, 373, 374, 375, 376, 377, 379, 385, 392, 394, 396, 403, 404, 410, 411, 413, 414, 417, 418, 422, 425, 426, 427, 428, 429, 430, 431, 432, 435, 436, 437, 439, 440, 441, 442, 445, 447, 449, 456, 458, 460, 461, 463, 466, 470, 471, 482, 483, 485, 486, 488, 492, 493, 494, 495, 496, 500, 501, 502, 504, 505, 509,

510, 511, 519, 520, 521, 525, 528, 529, 530, 531, 532, 533, 534, 536, 541, 542, 551, 552, 553, 554, 555, 558, 563, 565, 568, 569, 570, 571, 578, 582, 583, 585, 587, 589, 590, 591, 592, 593, 594, 595, 596, 599, 601, 603, 604, 605, 609, 612, 617, 619, 621, 628, 629, 630, 637, 638, 639, 640, 641, 642, 649, 650, 652, 654, 658, 659, 660, 667, 670, 672, 674, 675, 676, 680, 681, 682, 683, 684, 685, 686, 690, 693, 694, 695, 696, 701, 705, 711, 712, 713, 719, 724, 725, 726, 728, 729, 734, 735, 738, 739, 743, 744, 745, 748, 751, 752, 753, 754, 755, 757, 759, 770, 772, 774, 775, 781, 783, 784, 785, 786, 790, 791, 793, 794, 795, 796, 798, 799, 801, 804, 805, 806, 807, 808, 809, 813, 814, 828, 829, 831, 833, 834, 836, 837, 841, 842, 843, 846, 847, 849, 851, 852, 853, 854, 855, 856, 857, 859, 860, 861, 865, 866, 869, 870, 873, 874, 875, 880, 884, 885, 887, 888, 891, 894, 897, 906, 907, 908, 909, 910, 911, 912, 917, 918, 920, 922, 923, 924, 925, 926, 927, 928, 929, 930, 931, 932, 933, 934, 935, 936, 937, 938, 939, 940, 941, 942, 945, 946, 947, 949, 953, 961, 964, 965, 968, 970, 971, 972, 977, 979, 983, 984, 985, 986, 987, 988, 994, 995, 996, 997, 1000, 1001, 1002, 1003, 1004, 1005, 1006, 1007, 1008, 1012, 1013, 1024, 1025, 1026, 1027, 1029, 1031, 1032, 1034, 1037, 1038, 1039, 1040, 1041, 1042, 1045, 1046, 1047, 1048, 1049, 1050, 1051, 1052, 1055, 1056, 1058, 1060, 1061, 1063, 1066, 1067, 1068, 1069, 1070, 1072, 1073, 1074, 1076, 1078, 1079, 1081, 1082, 1083, 1084, 1085, 1088, 1089, 1090, 1094, 1095, 1096, 1100, 1105, 1106, 1108, 1111, 1112, 1113, 1114, 1115, 1118, 1119, 1120, 1121, 1124, 1128, 1129, 1133, 1134, 1135, 1136, 1137, 1138, 1139, 1140, 1141, 1142, 1143, 1144, 1145, 1147, 1151, 1152, 1153, 1156, 1158, 1160, 1164, 1165, 1166, 1167, 1169, 1173, 1174, 1176, 1179, 1184, 1185, 1193, 1195, 1196, 1197, 1198, 1200, 1201, 1202, 1203, 1205, 1207, 1219, 1222, 1223, 1224, 1226, 1233, 1234, 1235, 1236, 1238, 1239, 1242, 1243, 1244, 1248, 1251, 1258, 1269, 1275, 1276, 1278, 1279, 1280, 1281, 1282, 1283, 1284, 1285, 1288, 1289, 1291, 1292, 1293, 1295, 1296, 1297, 1298, 1303, 1304, 1306, 1309, 1313, 1314, 1316, 1317, 1322, 1323, 1324, 1325, 1333, 1334, 1335, 1337, 1338, 1339, 1340, 1341, 1345, 1346, 1348, 1353, 1354, 1355, 1356, 1357, 1358, 1360, 1361, 1368, 1371, 1372, 1377, 1378, 1380, 1383, 1385, 1387, 1395, 1399, 1400,

1402, 1403, 1406, 1407, 1408, 1414, 1415, 1423, 1425, 1426, 1427, 1428, 1429, 1430, 1432, 1434, 1436, 1440, 1441, 1445, 1448, 1453, 1455, 1458, 1459, 1460, 1466, 1474, 1476, 1477, 1481, 1482, 1483, 1484, 1485, 1489, 1490, 1492, 1495, 1496, 1498, 1499, 1500

25. **psychot. humans** humans, psychotic or labile
75, 170, 239, 245, 282, 285, 363, 398, 432, 517, 518, 535, 620, 773, 877, 884, 916, 931, 932, 1067, 1106, 1127, 1129, 1251, 1306, 1352, 1428, 1470, 1498
26. **drug-dep. humans** humans, drug-dependent or drug-dependence-prone
9, 11, 12, 19, 20, 21, 26, 38, 46, 63, 71, 82, 91, 97, 123, 127, 135, 139, 140, 146, 152, 153, 161, 162, 179, 196, 204, 249, 274, 277, 282, 284, 307, 308, 314, 315, 320, 321, 325, 347, 351, 354, 379, 393, 402, 411, 412, 416, 443, 454, 484, 503, 505, 507, 516, 517, 518, 523, 530, 535, 545, 550, 582, 586, 595, 620, 636, 655, 656, 657, 658, 665, 668, 669, 688, 691, 708, 717, 718, 719, 731, 732, 733, 743, 758, 767, 774, 786, 802, 803, 810, 821, 850, 851, 861, 864, 871, 872, 876, 893, 906, 907, 908, 909, 914, 915, 916, 933, 943, 952, 956, 958, 959, 966, 967, 983, 985, 1023, 1067, 1068, 1071, 1075, 1085, 1106, 1109, 1136, 1137, 1138, 1146, 1148, 1154, 1155, 1160, 1171, 1184, 1207, 1223, 1236, 1237, 1251, 1258, 1259, 1261, 1262, 1263, 1265, 1268, 1275, 1288, 1306, 1309, 1314, 1319, 1326, 1347, 1351, 1365, 1366, 1368, 1369, 1370, 1392, 1406, 1423, 1469, 1471, 1473, 1478, 1492
27. **mammals** mammals (excluding humans)
2, 3, 4, 5, 7, 8, 10, 14, 15, 16, 17, 18, 25, 27, 28, 29, 31, 34, 35, 54, 60, 76, 77, 78, 79, 80, 81, 84, 86, 88, 89, 90, 92, 93, 94, 98, 101, 102, 103, 109, 110, 111, 113, 115, 116, 119, 122, 125, 126, 140, 141, 142, 143, 149, 150, 151, 156, 159, 160, 163, 166, 167, 173, 176, 177, 181, 183, 184, 187, 190, 191, 192, 194, 196, 197, 201, 203, 205, 211, 215, 216, 217, 218, 219, 220, 221, 222, 223, 224, 225, 226, 227, 229, 232, 233, 234, 236, 241, 242, 243, 247, 248, 254, 257, 258, 259, 261, 262, 263, 264, 265, 266, 267, 269, 272, 273, 275, 278, 279, 280, 281, 287, 291, 292, 294, 295, 297, 299, 300, 304, 305, 311, 313, 316, 317, 318, 323, 334, 341, 345, 346, 355, 356, 357, 360, 364, 365, 366, 378, 381, 382, 383, 384, 386, 387, 388, 389, 390, 391, 395, 397, 399, 400, 401, 405, 406, 407, 408, 409, 415, 419, 420, 421, 423, 424, 426, 429, 430, 431, 432, 433, 434,

438, 446, 448, 450, 451, 452, 453, 457, 459,
 464, 465, 467, 468, 469, 472, 473, 474, 475,
 476, 477, 478, 479, 480, 481, 485, 486, 487,
 488, 489, 490, 491, 497, 498, 499, 500, 505,
 506, 508, 511, 512, 513, 514, 515, 520, 521,
 522, 524, 526, 537, 538, 539, 540, 542, 543,
 544, 548, 549, 557, 559, 560, 561, 562, 564,
 566, 567, 570, 572, 573, 574, 575, 576, 577,
 578, 579, 580, 581, 584, 588, 591, 597, 598,
 600, 602, 607, 608, 610, 613, 614, 615, 616,
 618, 622, 623, 624, 625, 626, 627, 631, 633,
 634, 643, 644, 645, 646, 647, 648, 651, 653,
 661, 662, 663, 664, 665, 666, 671, 674, 676,
 677, 678, 683, 687, 689, 692, 695, 697, 698,
 699, 700, 701, 702, 703, 704, 706, 707, 709,
 710, 714, 715, 716, 717, 718, 719, 720, 721,
 722, 723, 726, 727, 730, 736, 737, 740, 741,
 742, 746, 749, 750, 755, 756, 760, 761, 762,
 763, 764, 765, 766, 768, 769, 771, 775, 776,
 777, 778, 780, 782, 783, 784, 785, 786, 787,
 788, 789, 790, 792, 797, 799, 800, 811, 812,
 815, 816, 817, 818, 819, 820, 822, 823, 824,
 825, 826, 827, 829, 830, 831, 833, 834, 835,
 838, 839, 840, 845, 848, 858, 862, 863, 867,
 868, 878, 879, 881, 882, 883, 886, 889, 890,
 892, 894, 895, 896, 898, 899, 900, 901, 902,
 903, 904, 905, 913, 919, 921, 928, 944, 948,
 950, 951, 954, 955, 957, 960, 961, 962, 963,
 969, 973, 974, 975, 978, 979, 980, 981, 982,
 983, 989, 990, 991, 992, 993, 998, 999, 1010,
 1011, 1014, 1015, 1016, 1017, 1018, 1019,
 1020, 1030, 1032, 1033, 1035, 1036, 1043,
 1044, 1053, 1054, 1059, 1062, 1063, 1064,
 1065, 1077, 1082, 1086, 1087, 1089, 1092,
 1093, 1096, 1098, 1099, 1100, 1101, 1102,
 1103, 1104, 1107, 1110, 1116, 1117, 1122,
 1123, 1124, 1125, 1128, 1130, 1131, 1132,
 1142, 1143, 1144, 1147, 1149, 1150, 1153,
 1156, 1157, 1158, 1159, 1161, 1162, 1163,
 1164, 1165, 1166, 1167, 1168, 1169, 1170,
 1172, 1178, 1180, 1182, 1183, 1186, 1187,
 1188, 1189, 1190, 1192, 1194, 1199, 1204,
 1206, 1208, 1209, 1210, 1211, 1212, 1213,
 1214, 1215, 1216, 1217, 1218, 1220, 1221,
 1225, 1227, 1228, 1229, 1230, 1231, 1232,
 1240, 1241, 1243, 1245, 1246, 1247, 1252,
 1253, 1254, 1255, 1256, 1257, 1260, 1264,
 1266, 1267, 1270, 1271, 1272, 1273, 1274,
 1277, 1279, 1280, 1281, 1283, 1284, 1285,
 1286, 1287, 1290, 1294, 1299, 1300, 1301,
 1302, 1305, 1307, 1308, 1310, 1312, 1313,
 1315, 1318, 1320, 1321, 1325, 1327, 1329,
 1330, 1331, 1332, 1336, 1342, 1343, 1344,
 1349, 1350, 1359, 1362, 1363, 1367, 1371,

1374, 1375, 1376, 1379, 1381, 1382, 1384,
 1386, 1388, 1389, 1390, 1391, 1393, 1394,
 1396, 1397, 1398, 1399, 1405, 1407, 1409,
 1410, 1411, 1412, 1413, 1415, 1416, 1417,
 1418, 1419, 1420, 1421, 1422, 1423, 1424,
 1433, 1436, 1437, 1438, 1439, 1442, 1443,
 1444, 1446, 1447, 1448, 1449, 1450, 1451,
 1452, 1454, 1456, 1457, 1462, 1463, 1464,
 1465, 1467, 1472, 1475, 1479, 1480, 1486,
 1487, 1488, 1491, 1494, 1495, 1496, 1497,
 1498

28. **other org.** other organisms

2, 110, 118, 138, 164, 173, 232, 322, 334, 380,
 431, 455, 457, 466, 489, 575, 611, 635, 719,
 779, 812, 976, 1057, 1091, 1097, 1175, 1177,
 1180, 1181, 1183, 1191, 1243, 1294, 1328,
 1373, 1423, 1431, 1461, 1493

DRUG ADMINISTRATION IN EXPERIMENTAL STUDIES

29. **acute admin.** acute administration

5, 7, 8, 16, 17, 19, 22, 25, 27, 28, 29, 31, 34,
 35, 36, 54, 59, 61, 76, 77, 78, 80, 81, 84, 85,
 87, 89, 90, 92, 93, 94, 98, 99, 100, 108, 109,
 110, 111, 113, 116, 117, 118, 119, 122, 129,
 130, 131, 135, 136, 137, 139, 140, 141, 143,
 144, 147, 148, 149, 150, 151, 156, 158, 159,
 160, 161, 163, 165, 167, 168, 171, 173, 174,
 176, 177, 178, 180, 181, 182, 183, 184, 186,
 188, 189, 190, 191, 192, 194, 196, 197, 198,
 200, 201, 203, 205, 206, 207, 211, 213, 214,
 215, 216, 217, 218, 219, 220, 221, 222, 224,
 225, 227, 231, 232, 233, 234, 235, 236, 237,
 238, 239, 241, 242, 243, 244, 247, 248, 253,
 254, 257, 258, 261, 262, 263, 264, 265, 266,
 272, 273, 275, 276, 278, 279, 280, 281, 286,
 288, 289, 291, 292, 293, 294, 295, 297, 299,
 300, 302, 304, 305, 306, 316, 317, 318, 319,
 322, 323, 324, 326, 327, 328, 329, 331, 334,
 335, 337, 338, 339, 340, 341, 344, 350, 355,
 356, 357, 360, 361, 364, 365, 366, 369, 370,
 371, 372, 373, 377, 378, 380, 381, 382, 384,
 386, 387, 388, 389, 390, 391, 394, 395, 396,
 397, 401, 406, 407, 408, 409, 414, 417, 419,
 421, 422, 423, 424, 425, 426, 427, 428, 432,
 433, 434, 438, 441, 444, 448, 449, 450, 451,
 452, 453, 454, 455, 459, 463, 464, 465, 467,
 468, 469, 472, 473, 474, 475, 476, 478, 479,
 480, 481, 485, 486, 487, 488, 489, 490, 491,
 492, 493, 494, 495, 496, 497, 501, 503, 505,
 506, 508, 509, 510, 512, 513, 514, 515, 519,
 524, 526, 528, 529, 533, 535, 537, 538, 539,
 540, 541, 542, 543, 544, 547, 548, 549, 556,

557, 559, 560, 561, 562, 564, 565, 566, 567, 569, 572, 573, 574, 575, 576, 577, 578, 579, 583, 584, 585, 587, 588, 596, 597, 598, 599, 600, 601, 602, 603, 604, 605, 607, 608, 609, 610, 612, 613, 614, 616, 617, 618, 621, 622, 623, 624, 625, 626, 627, 629, 631, 633, 637, 640, 641, 643, 644, 645, 646, 647, 648, 650, 651, 652, 653, 656, 657, 658, 659, 660, 661, 662, 663, 664, 665, 666, 672, 676, 677, 678, 679, 681, 682, 684, 685, 686, 689, 692, 697, 698, 699, 700, 701, 702, 704, 706, 707, 709, 713, 714, 715, 716, 717, 718, 719, 720, 721, 722, 723, 726, 727, 730, 732, 733, 736, 737, 738, 740, 742, 745, 746, 748, 749, 750, 751, 752, 754, 755, 756, 760, 761, 762, 763, 764, 765, 766, 768, 769, 771, 774, 776, 777, 780, 782, 787, 788, 789, 792, 797, 798, 799, 800, 804, 805, 806, 811, 812, 815, 816, 817, 818, 819, 820, 822, 823, 824, 825, 826, 827, 828, 829, 830, 831, 833, 835, 836, 837, 841, 843, 845, 847, 848, 854, 858, 862, 863, 867, 868, 870, 871, 872, 873, 874, 875, 876, 877, 878, 879, 881, 882, 883, 886, 888, 889, 890, 891, 892, 893, 894, 895, 896, 898, 899, 900, 901, 902, 903, 904, 905, 911, 913, 921, 923, 927, 930, 931, 932, 933, 934, 935, 936, 937, 938, 939, 940, 941, 942, 944, 945, 946, 947, 948, 950, 951, 954, 955, 957, 961, 962, 963, 973, 974, 975, 979, 981, 983, 984, 988, 989, 990, 991, 992, 993, 995, 996, 997, 999, 1010, 1011, 1012, 1014, 1015, 1016, 1017, 1018, 1019, 1020, 1024, 1025, 1026, 1027, 1030, 1033, 1036, 1038, 1039, 1040, 1042, 1044, 1046, 1047, 1051, 1054, 1055, 1059, 1062, 1064, 1065, 1073, 1074, 1077, 1078, 1082, 1083, 1086, 1087, 1088, 1089, 1090, 1092, 1093, 1095, 1097, 1099, 1102, 1103, 1104, 1105, 1106, 1107, 1109, 1110, 1112, 1113, 1114, 1116, 1117, 1121, 1122, 1123, 1125, 1127, 1128, 1132, 1142, 1143, 1144, 1145, 1147, 1148, 1149, 1150, 1151, 1152, 1153, 1157, 1158, 1159, 1163, 1164, 1165, 1166, 1168, 1169, 1172, 1173, 1174, 1176, 1177, 1178, 1179, 1181, 1182, 1183, 1185, 1188, 1189, 1190, 1191, 1192, 1196, 1198, 1199, 1200, 1204, 1206, 1207, 1208, 1209, 1210, 1211, 1212, 1213, 1214, 1216, 1217, 1218, 1220, 1221, 1224, 1225, 1227, 1228, 1229, 1230, 1231, 1232, 1233, 1234, 1240, 1241, 1244, 1245, 1246, 1247, 1248, 1252, 1253, 1254, 1255, 1256, 1257, 1264, 1266, 1267, 1268, 1270, 1271, 1272, 1273, 1286, 1287, 1290, 1291, 1292, 1294, 1296, 1299, 1300, 1301, 1304, 1305, 1307, 1310, 1312, 1315, 1316,

1317, 1318, 1319, 1320, 1321, 1324, 1326, 1327, 1328, 1329, 1331, 1332, 1333, 1334, 1336, 1337, 1339, 1340, 1341, 1342, 1343, 1344, 1349, 1350, 1354, 1356, 1359, 1364, 1367, 1369, 1373, 1375, 1376, 1379, 1381, 1382, 1383, 1384, 1386, 1388, 1389, 1390, 1391, 1392, 1394, 1396, 1397, 1398, 1399, 1405, 1407, 1409, 1410, 1412, 1413, 1415, 1418, 1419, 1420, 1422, 1424, 1425, 1426, 1427, 1428, 1436, 1437, 1439, 1442, 1443, 1444, 1445, 1446, 1447, 1449, 1452, 1453, 1454, 1455, 1456, 1457, 1458, 1459, 1460, 1461, 1462, 1463, 1464, 1465, 1466, 1467, 1472, 1474, 1476, 1478, 1479, 1480, 1481, 1482, 1483, 1486, 1487, 1488, 1494, 1495, 1496, 1499, 1500

30. **chronic admin.** chronic administration

4, 8, 14, 15, 17, 18, 79, 80, 82, 88, 90, 101, 102, 103, 113, 114, 129, 142, 144, 146, 167, 168, 187, 201, 209, 210, 214, 221, 222, 231, 235, 239, 242, 246, 247, 257, 261, 286, 287, 303, 306, 311, 313, 318, 319, 378, 383, 400, 419, 420, 438, 443, 459, 466, 487, 530, 561, 568, 580, 610, 615, 617, 624, 637, 643, 645, 646, 656, 658, 666, 677, 687, 704, 707, 714, 719, 720, 736, 738, 741, 757, 762, 766, 768, 776, 778, 779, 787, 788, 819, 838, 840, 871, 872, 876, 889, 893, 894, 911, 919, 923, 944, 954, 957, 962, 963, 967, 969, 978, 979, 982, 983, 999, 1010, 1035, 1041, 1043, 1044, 1086, 1087, 1093, 1098, 1101, 1110, 1114, 1117, 1156, 1159, 1161, 1162, 1163, 1164, 1165, 1167, 1172, 1186, 1187, 1192, 1194, 1195, 1221, 1232, 1237, 1248, 1253, 1255, 1260, 1264, 1299, 1301, 1302, 1305, 1306, 1330, 1340, 1341, 1350, 1375, 1379, 1382, 1384, 1391, 1392, 1410, 1411, 1428, 1438, 1469, 1475, 1479, 1480, 1491, 1497

31. **in vivo** in vivo

4, 7, 8, 10, 14, 15, 16, 18, 19, 22, 25, 27, 28, 29, 31, 34, 35, 36, 54, 59, 61, 76, 77, 78, 79, 80, 81, 82, 84, 85, 87, 88, 89, 90, 92, 94, 98, 99, 100, 101, 102, 103, 108, 109, 110, 111, 113, 114, 116, 117, 118, 119, 125, 126, 129, 130, 131, 135, 136, 137, 138, 139, 140, 141, 142, 143, 144, 146, 147, 148, 149, 150, 151, 156, 158, 159, 160, 161, 163, 164, 165, 167, 171, 173, 174, 176, 177, 178, 180, 181, 182, 183, 184, 186, 187, 188, 189, 190, 191, 192, 194, 196, 197, 198, 200, 201, 203, 205, 206, 207, 209, 210, 211, 212, 213, 214, 215, 216, 217, 218, 219, 220, 221, 222, 224, 225, 227, 232, 233, 234, 235, 236, 237, 238, 239, 241, 242, 243, 244, 246, 247, 248, 253, 254, 257,

258, 261, 262, 263, 264, 265, 266, 272, 273,
 275, 276, 278, 279, 280, 284, 286, 287, 288,
 289, 291, 292, 293, 294, 295, 297, 300, 302,
 303, 304, 305, 306, 311, 313, 316, 317, 318,
 319, 323, 324, 326, 327, 328, 329, 331, 334,
 335, 337, 338, 339, 340, 341, 344, 350, 355,
 356, 357, 360, 361, 364, 365, 366, 369, 370,
 371, 372, 373, 377, 378, 380, 381, 382, 383,
 384, 386, 387, 388, 389, 390, 391, 394, 395,
 396, 397, 400, 401, 406, 407, 408, 409, 414,
 417, 419, 420, 421, 422, 423, 424, 425, 426,
 427, 428, 432, 433, 434, 438, 441, 443, 444,
 448, 449, 450, 451, 452, 453, 454, 455, 459,
 463, 464, 465, 466, 467, 468, 469, 472, 473,
 474, 475, 476, 478, 479, 480, 481, 485, 486,
 487, 488, 489, 490, 491, 492, 493, 494, 495,
 496, 497, 501, 503, 505, 506, 508, 509, 510,
 512, 513, 514, 515, 516, 524, 526, 528, 529,
 530, 533, 535, 537, 538, 539, 540, 541, 542,
 543, 544, 547, 548, 549, 556, 557, 559, 560,
 561, 562, 564, 565, 566, 567, 568, 569, 572,
 573, 574, 575, 576, 577, 578, 579, 582, 583,
 584, 585, 587, 588, 596, 597, 598, 599, 600,
 601, 602, 603, 604, 605, 607, 608, 609, 610,
 611, 612, 613, 614, 615, 616, 617, 618, 621,
 622, 623, 624, 625, 626, 627, 629, 631, 633,
 635, 637, 640, 641, 643, 644, 646, 647, 650,
 651, 652, 653, 656, 657, 658, 659, 660, 661,
 662, 663, 664, 665, 666, 672, 676, 677, 678,
 681, 682, 684, 685, 686, 687, 689, 692, 697,
 698, 699, 700, 701, 702, 703, 704, 706, 707,
 709, 713, 715, 716, 717, 718, 719, 720, 721,
 722, 723, 726, 727, 730, 732, 733, 736, 737,
 738, 740, 741, 742, 745, 746, 748, 749, 750,
 751, 752, 754, 755, 757, 760, 761, 762, 763,
 764, 765, 766, 768, 771, 774, 775, 776, 777,
 778, 779, 780, 782, 787, 788, 792, 796, 797,
 798, 799, 800, 804, 805, 806, 811, 812, 815,
 816, 817, 818, 819, 820, 822, 823, 824, 825,
 826, 827, 828, 829, 830, 831, 833, 835, 836,
 837, 838, 840, 841, 843, 845, 847, 848, 854,
 858, 862, 863, 867, 868, 870, 871, 872, 873,
 874, 875, 876, 877, 878, 879, 881, 882, 883,
 886, 888, 889, 890, 891, 892, 893, 894, 895,
 896, 898, 899, 900, 901, 902, 903, 904, 905,
 911, 913, 919, 921, 923, 927, 930, 931, 932,
 933, 934, 935, 936, 937, 938, 939, 940, 941,
 942, 944, 945, 946, 947, 948, 950, 951, 954,
 955, 957, 961, 962, 963, 967, 969, 973, 974,
 975, 978, 979, 981, 982, 983, 984, 989, 990,
 991, 992, 993, 995, 996, 997, 999, 1010, 1011,
 1012, 1014, 1015, 1016, 1017, 1019, 1020,
 1024, 1025, 1026, 1027, 1030, 1033, 1035,
 1036, 1038, 1039, 1040, 1041, 1042, 1043,

1044, 1046, 1047, 1051, 1054, 1055, 1059,
 1062, 1065, 1073, 1074, 1077, 1078, 1082,
 1083, 1084, 1086, 1087, 1088, 1089, 1090,
 1092, 1093, 1095, 1097, 1098, 1099, 1101,
 1102, 1103, 1104, 1105, 1106, 1107, 1109,
 1110, 1112, 1113, 1116, 1117, 1121, 1122,
 1123, 1125, 1127, 1128, 1131, 1132, 1142,
 1143, 1144, 1145, 1147, 1148, 1149, 1150,
 1151, 1152, 1153, 1156, 1157, 1158, 1159,
 1161, 1162, 1164, 1165, 1166, 1167, 1168,
 1169, 1172, 1173, 1174, 1176, 1177, 1178,
 1179, 1182, 1183, 1185, 1186, 1187, 1188,
 1192, 1194, 1195, 1196, 1198, 1199, 1200,
 1204, 1206, 1207, 1208, 1209, 1210, 1211,
 1212, 1213, 1214, 1216, 1217, 1218, 1221,
 1224, 1225, 1227, 1228, 1229, 1230, 1231,
 1232, 1233, 1234, 1237, 1240, 1241, 1244,
 1245, 1246, 1247, 1248, 1252, 1253, 1254,
 1255, 1256, 1257, 1260, 1264, 1266, 1267,
 1268, 1270, 1271, 1272, 1273, 1286, 1287,
 1290, 1291, 1292, 1294, 1296, 1299, 1300,
 1301, 1302, 1304, 1305, 1306, 1307, 1310,
 1312, 1315, 1316, 1317, 1318, 1319, 1320,
 1321, 1324, 1326, 1327, 1328, 1329, 1331,
 1332, 1333, 1334, 1336, 1337, 1339, 1340,
 1341, 1342, 1343, 1349, 1350, 1354, 1356,
 1359, 1364, 1367, 1369, 1373, 1375, 1376,
 1379, 1382, 1383, 1384, 1386, 1388, 1389,
 1390, 1391, 1392, 1394, 1396, 1397, 1398,
 1399, 1405, 1407, 1409, 1410, 1411, 1412,
 1413, 1415, 1416, 1419, 1420, 1422, 1424,
 1425, 1426, 1427, 1428, 1436, 1437, 1438,
 1439, 1442, 1443, 1444, 1445, 1446, 1447,
 1449, 1452, 1453, 1454, 1455, 1456, 1457,
 1458, 1459, 1460, 1461, 1462, 1463, 1464,
 1465, 1466, 1467, 1469, 1472, 1474, 1475,
 1476, 1478, 1481, 1482, 1483, 1486, 1487,
 1488, 1491, 1494, 1495, 1496, 1497, 1499,
 1500

32. *in vitro* in vitro

5, 17, 110, 111, 122, 125, 190, 232, 257, 266,
 299, 322, 366, 399, 405, 415, 433, 477, 497,
 519, 522, 560, 576, 580, 581, 582, 626, 634,
 645, 646, 648, 662, 663, 671, 678, 679, 703,
 704, 707, 710, 719, 727, 769, 775, 788, 789,
 799, 827, 828, 840, 894, 962, 973, 976, 979,
 980, 983, 998, 1018, 1033, 1057, 1064, 1091,
 1099, 1110, 1116, 1117, 1130, 1131, 1142,
 1143, 1144, 1163, 1164, 1166, 1167, 1168,
 1170, 1180, 1181, 1189, 1190, 1191, 1210,
 1220, 1271, 1272, 1273, 1308, 1325, 1330,
 1344, 1362, 1363, 1381, 1384, 1393, 1417,
 1418, 1419, 1421, 1431, 1433, 1436, 1450,
 1451, 1465, 1480, 1488, 1493, 1495

RESULTS OF DRUG INTAKE IN
EXPERIMENTAL AND
NON-EXPERIMENTAL PAPERS

33. **dose resp.** dose response curve

2, 8, 25, 31, 47, 52, 58, 64, 77, 92, 93, 94, 99,
109, 110, 147, 148, 159, 164, 184, 194, 197,
214, 220, 221, 222, 243, 247, 261, 274, 278,
295, 303, 305, 316, 323, 337, 355, 356, 357,
365, 370, 380, 381, 384, 388, 389, 395, 397,
401, 405, 423, 424, 438, 452, 467, 468, 472,
474, 475, 479, 481, 485, 486, 487, 488, 489,
490, 491, 497, 505, 506, 508, 522, 524, 526,
537, 539, 541, 564, 574, 577, 578, 579, 582,
598, 599, 602, 613, 623, 633, 634, 635, 645,
653, 679, 697, 704, 714, 721, 723, 730, 741,
744, 756, 762, 771, 776, 778, 792, 804, 812,
823, 826, 830, 831, 833, 836, 837, 852, 863,
867, 883, 891, 896, 898, 899, 900, 904, 905,
913, 946, 950, 957, 962, 963, 969, 972, 975,
983, 991, 999, 1014, 1019, 1036, 1053, 1054,
1059, 1084, 1089, 1090, 1107, 1147, 1149,
1170, 1175, 1178, 1180, 1181, 1182, 1188,
1191, 1204, 1207, 1221, 1228, 1240, 1241,
1246, 1252, 1254, 1255, 1267, 1273, 1290,
1294, 1299, 1300, 1312, 1315, 1318, 1321,
1327, 1329, 1350, 1359, 1382, 1391, 1396,
1399, 1407, 1412, 1418, 1420, 1421, 1424,
1442, 1444, 1446, 1449, 1452, 1454, 1463,
1473

34. **blood lev.** levels of drugs in blood

5, 10, 12, 13, 14, 16, 17, 22, 23, 24, 27, 37,
43, 47, 52, 54, 59, 61, 65, 78, 82, 85, 87, 92,
94, 99, 100, 105, 106, 108, 114, 117, 119, 121,
124, 125, 128, 129, 130, 131, 133, 136, 137,
144, 147, 152, 155, 158, 161, 172, 174, 175,
181, 182, 184, 186, 187, 188, 189, 190, 191,
192, 196, 198, 199, 200, 209, 213, 214, 215,
216, 221, 222, 228, 231, 232, 233, 234, 235,
236, 239, 242, 244, 246, 249, 251, 257, 258,
261, 262, 263, 264, 279, 281, 283, 286, 293,
294, 298, 303, 305, 306, 308, 316, 318, 323,
326, 337, 338, 339, 340, 342, 343, 348, 358,
359, 360, 364, 365, 366, 368, 369, 370, 371,
372, 373, 375, 376, 377, 379, 381, 382, 384,
385, 390, 393, 394, 397, 399, 403, 406, 409,
411, 414, 417, 419, 420, 422, 425, 427, 428,
429, 430, 431, 432, 434, 441, 443, 449, 451,
452, 460, 463, 467, 468, 476, 480, 481, 485,
486, 488, 492, 493, 494, 495, 496, 497, 498,
499, 505, 509, 510, 511, 512, 513, 514, 520,
521, 526, 528, 529, 530, 531, 533, 534, 535,
536, 538, 540, 541, 542, 543, 544, 552, 556,
557, 561, 564, 567, 570, 571, 582, 583, 584,

586, 591, 596, 597, 598, 599, 600, 601, 602,
603, 604, 605, 607, 608, 614, 617, 618, 619,
620, 623, 626, 629, 631, 632, 633, 640, 643,
646, 647, 649, 650, 652, 655, 656, 657, 658,
659, 660, 662, 663, 664, 665, 673, 674, 676,
677, 678, 681, 682, 683, 684, 685, 686, 689,
692, 693, 695, 697, 701, 703, 704, 707, 713,
717, 718, 721, 722, 724, 734, 736, 739, 742,
745, 746, 749, 750, 751, 752, 754, 755, 757,
758, 759, 768, 774, 776, 787, 788, 794, 799,
804, 805, 806, 807, 815, 824, 825, 826, 827,
828, 829, 833, 837, 839, 847, 848, 854, 855,
858, 862, 863, 869, 870, 873, 877, 891, 892,
893, 894, 905, 907, 908, 909, 910, 919, 921,
923, 924, 925, 926, 927, 932, 933, 934, 936,
937, 945, 946, 947, 950, 962, 963, 965, 967,
971, 973, 974, 981, 982, 983, 984, 987, 988,
995, 996, 999, 1001, 1002, 1003, 1005, 1006,
1008, 1011, 1015, 1021, 1023, 1024, 1025,
1026, 1027, 1028, 1029, 1033, 1036, 1040,
1042, 1043, 1046, 1047, 1055, 1058, 1065,
1066, 1067, 1070, 1073, 1074, 1076, 1082,
1084, 1089, 1090, 1093, 1099, 1100, 1103,
1110, 1111, 1112, 1113, 1117, 1118, 1122,
1125, 1126, 1127, 1128, 1136, 1137, 1138,
1139, 1140, 1141, 1142, 1145, 1146, 1147,
1148, 1150, 1166, 1169, 1174, 1175, 1177,
1178, 1179, 1184, 1185, 1195, 1196, 1197,
1198, 1199, 1200, 1201, 1203, 1206, 1207,
1208, 1210, 1211, 1213, 1214, 1215, 1221,
1224, 1225, 1228, 1229, 1230, 1231, 1232,
1233, 1241, 1244, 1245, 1246, 1248, 1256,
1257, 1258, 1259, 1270, 1271, 1272, 1273,
1276, 1280, 1281, 1282, 1283, 1284, 1287,
1291, 1295, 1297, 1298, 1310, 1313, 1322,
1323, 1326, 1333, 1334, 1335, 1336, 1337,
1338, 1346, 1349, 1353, 1355, 1356, 1358,
1367, 1369, 1383, 1388, 1389, 1397, 1398,
1399, 1401, 1405, 1406, 1407, 1420, 1424,
1425, 1426, 1427, 1428, 1429, 1430, 1432,
1440, 1444, 1445, 1453, 1455, 1458, 1462,
1463, 1464, 1465, 1466, 1467, 1469, 1471,
1472, 1475, 1476, 1481, 1483, 1486, 1487,
1488, 1490, 1497, 1498, 1499, 1500

35. **other drug lev.** levels of drugs, other than in
blood (urine, breath, sweat, saliva, faecal
levels)

14, 36, 37, 43, 68, 72, 83, 89, 90, 93, 111, 113,
114, 120, 124, 125, 131, 148, 149, 155, 158,
160, 164, 176, 183, 188, 190, 191, 192, 196,
198, 213, 228, 232, 234, 244, 249, 251, 261,
270, 280, 281, 294, 311, 358, 373, 379, 382,
390, 414, 417, 425, 427, 428, 431, 432, 493,
505, 509, 530, 532, 534, 541, 557, 558, 559,

- 566, 576, 582, 591, 603, 605, 619, 622, 632, 633, 649, 650, 651, 653, 659, 660, 662, 663, 672, 676, 697, 713, 717, 736, 745, 750, 751, 755, 779, 824, 833, 836, 842, 855, 865, 869, 870, 888, 893, 907, 908, 909, 925, 936, 945, 951, 982, 983, 995, 999, 1015, 1021, 1025, 1026, 1027, 1042, 1053, 1055, 1062, 1067, 1093, 1096, 1127, 1137, 1138, 1141, 1149, 1150, 1184, 1201, 1202, 1211, 1213, 1214, 1225, 1231, 1234, 1240, 1263, 1265, 1283, 1305, 1322, 1323, 1329, 1337, 1340, 1341, 1353, 1363, 1367, 1369, 1384, 1388, 1389, 1399, 1401, 1463, 1465, 1466, 1469, 1476, 1486, 1495, 1497, 1499, 1500
36. **mot. perform.** motor performance
9, 28, 59, 60, 63, 70, 82, 83, 86, 98, 105, 117, 126, 131, 136, 137, 139, 140, 144, 151, 166, 168, 174, 180, 182, 186, 188, 189, 206, 212, 214, 215, 244, 276, 284, 291, 292, 293, 294, 326, 327, 328, 331, 336, 344, 361, 363, 370, 371, 373, 375, 396, 403, 413, 422, 423, 426, 429, 430, 431, 432, 434, 441, 448, 453, 454, 463, 468, 469, 491, 492, 493, 494, 495, 496, 497, 511, 516, 531, 544, 556, 561, 572, 582, 591, 604, 605, 606, 609, 620, 629, 636, 637, 641, 646, 648, 659, 660, 666, 673, 681, 682, 684, 685, 686, 694, 701, 722, 724, 732, 733, 751, 754, 780, 816, 836, 837, 843, 873, 874, 875, 881, 883, 888, 902, 921, 923, 925, 929, 934, 935, 942, 945, 946, 964, 978, 984, 993, 1039, 1042, 1051, 1059, 1062, 1097, 1099, 1100, 1108, 1113, 1114, 1160, 1169, 1174, 1176, 1195, 1196, 1197, 1207, 1232, 1270, 1279, 1283, 1291, 1292, 1304, 1315, 1316, 1317, 1333, 1334, 1337, 1339, 1346, 1356, 1387, 1390, 1394, 1406, 1414, 1419, 1420, 1422, 1427, 1441, 1453, 1456, 1458, 1473, 1474, 1476, 1486, 1498, 1499
37. **psychol. perform.** psychological performance
27, 28, 59, 60, 63, 68, 82, 117, 131, 135, 136, 137, 139, 144, 182, 184, 186, 188, 206, 214, 244, 274, 297, 321, 327, 329, 330, 331, 335, 350, 363, 370, 372, 373, 375, 377, 396, 398, 422, 424, 425, 426, 427, 428, 429, 430, 431, 432, 441, 453, 454, 461, 491, 493, 494, 495, 496, 511, 515, 516, 518, 556, 572, 582, 586, 591, 597, 598, 599, 600, 601, 602, 603, 604, 605, 612, 629, 636, 637, 640, 641, 650, 659, 660, 673, 681, 682, 691, 701, 751, 754, 796, 801, 836, 837, 841, 843, 861, 870, 873, 877, 903, 916, 924, 925, 927, 929, 931, 932, 938, 942, 944, 945, 946, 953, 964, 967, 989, 990, 1009, 1042, 1051, 1059, 1067, 1078, 1084, 1097, 1100, 1106, 1109, 1112, 1113, 1114, 1157, 1169, 1177, 1195, 1196, 1201, 1237, 1285, 1288, 1291, 1292, 1296, 1304, 1333, 1334, 1337, 1339, 1372, 1379, 1394, 1406, 1414, 1427, 1428, 1434, 1441, 1449, 1458, 1473, 1474, 1476, 1498, 1499, 1500
38. **species or sex diff.** species or sex differences in response
8, 86, 92, 110, 149, 160, 163, 183, 220, 236, 269, 294, 297, 311, 317, 401, 404, 428, 432, 459, 489, 526, 561, 600, 608, 614, 676, 787, 788, 835, 840, 843, 854, 916, 919, 929, 931, 932, 973, 1051, 1067, 1097, 1099, 1100, 1113, 1142, 1143, 1144, 1182, 1199, 1271, 1272, 1280, 1281, 1284, 1325, 1343, 1437, 1491, 1498
- ANATOMICAL COMPONENTS OR
PHYSIOLOGICAL PROCESSES AFFECTED
BY INTERACTION**
39. **absorp., distrib., stor.** absorption, distribution, or storage
34, 54, 62, 64, 67, 92, 94, 96, 125, 136, 137, 140, 144, 158, 160, 161, 164, 167, 172, 176, 187, 192, 211, 216, 232, 233, 234, 244, 254, 262, 269, 270, 281, 287, 295, 305, 309, 315, 317, 343, 345, 349, 362, 368, 374, 405, 417, 431, 446, 455, 468, 470, 474, 493, 497, 500, 505, 514, 519, 522, 526, 532, 542, 546, 564, 565, 576, 583, 607, 608, 611, 620, 622, 626, 631, 643, 646, 653, 663, 677, 690, 691, 727, 734, 736, 740, 746, 751, 755, 758, 774, 779, 783, 784, 825, 826, 827, 828, 839, 858, 863, 897, 905, 925, 932, 943, 945, 977, 982, 983, 988, 995, 1015, 1020, 1021, 1031, 1034, 1037, 1044, 1049, 1050, 1061, 1073, 1074, 1084, 1090, 1093, 1102, 1107, 1124, 1126, 1127, 1128, 1132, 1136, 1137, 1138, 1141, 1145, 1166, 1168, 1201, 1205, 1213, 1225, 1227, 1236, 1240, 1241, 1260, 1263, 1280, 1281, 1283, 1284, 1287, 1293, 1304, 1305, 1309, 1329, 1338, 1349, 1380, 1381, 1389, 1413, 1415, 1423, 1425, 1426, 1439, 1454, 1463, 1465, 1466, 1481, 1483, 1486, 1497, 1498
40. **acid-base, blood pH, elect.** acid-base balance, blood pH, electrolytes
1, 11, 12, 13, 101, 114, 120, 125, 156, 163, 164, 169, 172, 244, 269, 345, 379, 392, 431, 476, 480, 481, 482, 493, 501, 532, 566, 582, 624, 718, 726, 742, 750, 766, 820, 827, 842, 864, 915, 1012, 1032, 1036, 1096, 1122, 1136, 1137, 1138, 1139, 1140, 1141, 1142, 1143, 1144, 1242, 1267, 1303, 1355, 1393, 1417, 1419, 1421, 1423, 1451, 1480, 1493

41. **blood comp., sites, lymph** blood components (blood cells, platelets, plasma), cell-forming sites, and lymph

26, 120, 139, 143, 181, 199, 228, 251, 265, 272, 273, 274, 295, 310, 311, 317, 318, 370, 379, 384, 394, 431, 433, 437, 439, 440, 446, 459, 465, 470, 480, 481, 482, 497, 501, 507, 519, 522, 532, 550, 551, 558, 582, 623, 624, 625, 627, 639, 653, 675, 697, 699, 704, 738, 740, 742, 743, 791, 829, 842, 846, 851, 864, 897, 912, 915, 920, 953, 956, 958, 959, 977, 1043, 1115, 1121, 1137, 1138, 1186, 1200, 1221, 1248, 1308, 1314, 1322, 1323, 1326, 1331, 1335, 1388, 1389, 1419, 1423, 1431, 1432

42. **cardiovasc.** cardiovascular system (heart, blood vessels, blood circulation)

1, 19, 26, 42, 58, 59, 60, 69, 70, 78, 82, 84, 88, 98, 106, 107, 109, 110, 127, 132, 134, 140, 141, 145, 146, 147, 152, 157, 160, 167, 171, 188, 193, 200, 202, 204, 208, 209, 215, 221, 222, 228, 237, 238, 243, 244, 245, 251, 252, 253, 259, 268, 288, 289, 296, 304, 311, 318, 319, 322, 324, 353, 359, 363, 370, 376, 379, 385, 390, 391, 392, 393, 394, 410, 414, 431, 437, 440, 446, 454, 455, 461, 464, 466, 470, 484, 486, 489, 490, 500, 516, 517, 520, 521, 523, 525, 527, 532, 536, 538, 544, 545, 550, 551, 555, 563, 568, 575, 579, 582, 590, 606, 607, 610, 620, 627, 636, 638, 639, 652, 667, 668, 669, 683, 688, 695, 702, 703, 705, 708, 711, 712, 718, 719, 721, 724, 725, 728, 729, 731, 732, 733, 744, 748, 750, 751, 753, 758, 766, 772, 774, 781, 782, 783, 784, 795, 796, 798, 802, 803, 808, 814, 818, 821, 823, 827, 837, 841, 842, 845, 846, 850, 851, 852, 856, 866, 868, 873, 877, 895, 897, 898, 899, 905, 911, 912, 915, 920, 922, 930, 933, 939, 940, 943, 949, 953, 954, 968, 975, 1013, 1016, 1020, 1029, 1030, 1034, 1036, 1037, 1038, 1043, 1057, 1059, 1078, 1081, 1088, 1091, 1100, 1103, 1113, 1115, 1119, 1121, 1127, 1128, 1133, 1135, 1136, 1137, 1138, 1149, 1151, 1152, 1153, 1156, 1157, 1158, 1159, 1172, 1173, 1183, 1184, 1187, 1191, 1193, 1207, 1222, 1235, 1238, 1240, 1241, 1246, 1248, 1263, 1283, 1294, 1324, 1326, 1331, 1340, 1341, 1342, 1343, 1346, 1350, 1357, 1369, 1370, 1371, 1374, 1387, 1395, 1406, 1423, 1443, 1444, 1453, 1465, 1472, 1478, 1480, 1483, 1492, 1493, 1497

43. **CNS** central nervous system

1, 2, 3, 8, 9, 14, 15, 18, 19, 27, 28, 30, 31, 32, 40, 42, 45, 49, 50, 53, 54, 56, 63, 68, 74, 76,

87, 93, 94, 99, 104, 105, 107, 110, 116, 120, 122, 123, 126, 127, 130, 133, 135, 136, 137, 139, 140, 141, 142, 144, 150, 151, 157, 159, 165, 167, 173, 176, 177, 180, 183, 184, 188, 189, 190, 193, 194, 195, 196, 200, 203, 204, 205, 206, 207, 209, 213, 214, 215, 217, 218, 219, 220, 221, 222, 224, 225, 226, 227, 228, 229, 236, 237, 244, 247, 248, 250, 251, 254, 258, 261, 262, 264, 269, 274, 275, 278, 285, 291, 294, 297, 298, 299, 302, 307, 312, 315, 320, 325, 328, 330, 336, 338, 339, 340, 341, 342, 343, 344, 346, 348, 349, 352, 353, 354, 356, 363, 364, 365, 370, 372, 373, 379, 388, 389, 391, 393, 394, 395, 405, 410, 413, 415, 421, 422, 423, 424, 425, 426, 427, 429, 430, 431, 432, 434, 435, 438, 439, 441, 443, 445, 446, 447, 449, 451, 452, 453, 459, 460, 463, 464, 466, 468, 469, 470, 471, 475, 478, 479, 481, 482, 483, 484, 489, 490, 491, 492, 493, 494, 495, 496, 497, 498, 499, 500, 503, 504, 505, 506, 508, 511, 513, 514, 515, 516, 517, 520, 521, 526, 527, 531, 536, 538, 540, 542, 545, 547, 548, 549, 554, 556, 559, 560, 561, 562, 569, 572, 573, 574, 576, 577, 581, 582, 585, 587, 588, 591, 592, 595, 597, 600, 601, 603, 609, 614, 615, 616, 620, 621, 627, 630, 633, 637, 640, 642, 644, 645, 646, 647, 651, 652, 653, 660, 661, 662, 665, 666, 667, 674, 676, 677, 680, 683, 685, 686, 688, 691, 694, 709, 714, 715, 716, 717, 718, 719, 720, 721, 722, 723, 724, 725, 728, 730, 732, 733, 736, 737, 745, 747, 754, 757, 758, 760, 762, 765, 766, 771, 773, 783, 784, 791, 792, 794, 797, 800, 801, 802, 803, 804, 808, 809, 811, 814, 816, 818, 819, 820, 822, 826, 828, 836, 837, 839, 848, 849, 850, 851, 852, 853, 856, 861, 862, 863, 867, 871, 872, 873, 876, 877, 878, 879, 880, 881, 882, 883, 884, 885, 887, 889, 890, 893, 897, 898, 899, 900, 902, 903, 904, 905, 913, 918, 920, 922, 923, 925, 928, 930, 933, 935, 936, 937, 938, 940, 941, 945, 946, 947, 948, 949, 950, 953, 954, 957, 958, 959, 961, 964, 965, 966, 967, 968, 978, 982, 983, 984, 985, 986, 992, 997, 998, 999, 1000, 1007, 1009, 1014, 1015, 1016, 1017, 1018, 1020, 1021, 1022, 1023, 1025, 1026, 1027, 1028, 1029, 1031, 1034, 1035, 1037, 1038, 1042, 1043, 1044, 1045, 1046, 1047, 1048, 1049, 1050, 1051, 1052, 1053, 1054, 1056, 1058, 1059, 1062, 1066, 1067, 1068, 1069, 1070, 1071, 1073, 1074, 1075, 1077, 1078, 1079, 1083, 1084, 1087, 1088, 1090, 1093, 1094, 1097, 1099, 1100, 1104, 1105, 1106, 1107, 1108, 1109, 1110, 1112, 1113, 1114, 1116,

- 1117, 1120, 1126, 1129, 1133, 1134, 1136, 1137, 1138, 1139, 1142, 1148, 1149, 1150, 1154, 1155, 1160, 1164, 1165, 1169, 1171, 1172, 1174, 1175, 1176, 1177, 1182, 1183, 1184, 1185, 1187, 1188, 1192, 1193, 1194, 1195, 1196, 1198, 1204, 1205, 1207, 1211, 1215, 1216, 1217, 1218, 1219, 1222, 1225, 1226, 1227, 1231, 1236, 1237, 1239, 1243, 1244, 1245, 1246, 1247, 1248, 1251, 1252, 1253, 1254, 1255, 1258, 1261, 1262, 1263, 1266, 1267, 1269, 1270, 1273, 1274, 1277, 1278, 1279, 1280, 1281, 1283, 1284, 1286, 1288, 1289, 1290, 1292, 1294, 1295, 1296, 1297, 1301, 1302, 1305, 1306, 1307, 1309, 1311, 1313, 1315, 1316, 1317, 1318, 1319, 1320, 1321, 1325, 1326, 1327, 1330, 1331, 1332, 1336, 1340, 1341, 1342, 1343, 1345, 1351, 1352, 1353, 1356, 1357, 1358, 1361, 1366, 1369, 1370, 1371, 1372, 1373, 1375, 1376, 1377, 1378, 1379, 1380, 1382, 1387, 1390, 1391, 1392, 1394, 1395, 1400, 1401, 1402, 1403, 1404, 1406, 1407, 1408, 1409, 1410, 1411, 1412, 1413, 1414, 1415, 1420, 1421, 1422, 1423, 1424, 1427, 1428, 1429, 1430, 1434, 1435, 1438, 1440, 1441, 1442, 1445, 1446, 1448, 1449, 1452, 1453, 1455, 1456, 1457, 1458, 1459, 1460, 1463, 1464, 1465, 1469, 1470, 1471, 1474, 1476, 1478, 1479, 1483, 1486, 1489, 1492, 1494, 1496, 1497, 1498
44. **G.I. tract** gastro-intestinal tract, its glands and secretions
1, 4, 30, 64, 70, 82, 91, 98, 110, 139, 140, 164, 184, 209, 240, 244, 250, 251, 262, 267, 270, 288, 289, 295, 324, 345, 352, 354, 355, 363, 379, 391, 393, 401, 410, 417, 431, 437, 440, 465, 470, 501, 503, 507, 517, 523, 527, 532, 542, 545, 551, 558, 564, 575, 590, 606, 621, 627, 632, 638, 667, 690, 691, 696, 697, 711, 719, 725, 727, 735, 740, 743, 748, 755, 758, 769, 770, 772, 783, 784, 798, 814, 825, 826, 827, 842, 846, 847, 850, 851, 854, 856, 885, 895, 915, 920, 937, 943, 951, 953, 968, 977, 983, 986, 994, 1012, 1016, 1020, 1027, 1032, 1038, 1049, 1085, 1094, 1100, 1115, 1126, 1127, 1128, 1132, 1133, 1134, 1135, 1136, 1137, 1138, 1150, 1153, 1170, 1171, 1207, 1225, 1243, 1248, 1260, 1263, 1265, 1287, 1309, 1316, 1317, 1353, 1388, 1389, 1413, 1423, 1438, 1439, 1451, 1454, 1492, 1494
45. **glands** glands (exocrine and endocrine) and their secretions, except items 44 and 46
34, 70, 88, 128, 132, 143, 178, 237, 276, 342, 419, 431, 527, 566, 580, 586, 688, 697, 719, 795, 796, 843, 850, 892, 915, 968, 1082, 1150, 1235, 1248, 1364, 1423, 1447, 1497
46. **liver, kidney** liver, kidney (including urine)
1, 4, 17, 26, 34, 35, 39, 50, 78, 80, 81, 88, 96, 111, 113, 114, 116, 120, 127, 139, 143, 157, 160, 161, 163, 168, 178, 181, 190, 191, 192, 198, 201, 223, 224, 228, 229, 231, 233, 234, 242, 243, 244, 245, 251, 257, 266, 270, 272, 274, 287, 299, 301, 303, 317, 318, 354, 359, 360, 361, 379, 383, 384, 391, 392, 393, 399, 400, 409, 417, 431, 432, 433, 434, 435, 437, 440, 444, 465, 468, 470, 474, 497, 507, 522, 526, 527, 530, 532, 541, 542, 545, 551, 556, 558, 559, 561, 566, 570, 575, 576, 580, 582, 583, 607, 616, 618, 621, 622, 626, 634, 636, 643, 656, 657, 658, 662, 663, 666, 667, 671, 675, 677, 687, 688, 697, 698, 699, 700, 704, 706, 707, 710, 712, 719, 736, 738, 740, 741, 742, 743, 750, 758, 766, 767, 768, 769, 770, 775, 776, 777, 778, 786, 787, 788, 789, 790, 792, 802, 803, 805, 806, 819, 821, 824, 840, 842, 846, 850, 857, 861, 864, 882, 886, 892, 894, 897, 898, 899, 901, 914, 915, 919, 920, 926, 943, 952, 953, 954, 955, 973, 974, 977, 978, 979, 980, 983, 999, 1011, 1033, 1037, 1043, 1055, 1064, 1069, 1085, 1089, 1093, 1102, 1110, 1121, 1124, 1125, 1130, 1133, 1135, 1137, 1138, 1142, 1150, 1161, 1162, 1163, 1164, 1165, 1166, 1167, 1168, 1184, 1186, 1187, 1189, 1190, 1206, 1211, 1215, 1216, 1217, 1218, 1220, 1224, 1231, 1236, 1254, 1255, 1257, 1263, 1265, 1271, 1272, 1273, 1275, 1289, 1309, 1311, 1312, 1338, 1344, 1353, 1360, 1362, 1365, 1381, 1384, 1386, 1389, 1395, 1423, 1432, 1433, 1436, 1437, 1440, 1446, 1447, 1450, 1462, 1464, 1465, 1475, 1483, 1486, 1487, 1488, 1491, 1492, 1495, 1497
47. **metab. proc.** metabolic processes
1, 4, 5, 10, 11, 12, 13, 16, 27, 35, 36, 45, 54, 61, 62, 80, 81, 85, 90, 98, 100, 105, 106, 107, 108, 111, 114, 116, 118, 121, 125, 129, 133, 140, 147, 148, 149, 151, 156, 158, 160, 161, 163, 166, 167, 168, 169, 176, 177, 178, 185, 195, 198, 199, 200, 201, 203, 205, 207, 208, 209, 211, 215, 216, 231, 232, 233, 234, 239, 241, 242, 244, 246, 251, 255, 257, 258, 260, 261, 262, 263, 264, 265, 266, 267, 269, 271, 272, 273, 274, 280, 281, 286, 289, 294, 299, 300, 303, 305, 309, 310, 311, 315, 316, 317, 323, 337, 341, 342, 349, 354, 360, 362, 368, 374, 376, 378, 379, 386, 391, 393, 394, 397, 399, 405, 406, 407, 408, 409, 414, 415, 417, 420, 431, 432, 433, 444, 446, 451, 457, 466,

- 473, 474, 477, 479, 480, 481, 482, 490, 495, 496, 497, 500, 511, 512, 513, 514, 520, 521, 522, 526, 528, 532, 533, 535, 536, 540, 542, 545, 547, 557, 561, 567, 569, 570, 580, 581, 582, 607, 614, 618, 621, 622, 626, 629, 631, 638, 643, 644, 646, 649, 651, 652, 653, 655, 656, 657, 658, 662, 663, 664, 671, 672, 676, 677, 678, 679, 687, 688, 689, 692, 697, 701, 703, 706, 707, 710, 716, 718, 719, 723, 724, 734, 737, 738, 746, 749, 750, 751, 753, 755, 766, 768, 769, 775, 776, 777, 782, 783, 784, 785, 786, 787, 788, 789, 790, 794, 805, 806, 807, 810, 818, 819, 824, 833, 834, 839, 840, 841, 848, 855, 866, 894, 900, 904, 911, 912, 919, 921, 939, 954, 955, 960, 962, 963, 965, 973, 979, 980, 983, 988, 996, 999, 1018, 1033, 1034, 1035, 1037, 1040, 1041, 1053, 1062, 1064, 1065, 1073, 1074, 1084, 1089, 1093, 1095, 1096, 1097, 1099, 1100, 1101, 1102, 1103, 1110, 1116, 1117, 1118, 1122, 1123, 1124, 1125, 1127, 1131, 1136, 1137, 1138, 1139, 1141, 1142, 1143, 1144, 1145, 1149, 1154, 1162, 1163, 1164, 1165, 1166, 1167, 1168, 1178, 1189, 1190, 1192, 1193, 1199, 1200, 1201, 1206, 1208, 1209, 1210, 1211, 1212, 1213, 1214, 1215, 1216, 1217, 1218, 1224, 1228, 1229, 1232, 1234, 1236, 1243, 1254, 1255, 1256, 1264, 1271, 1272, 1273, 1276, 1279, 1280, 1281, 1283, 1284, 1285, 1288, 1289, 1293, 1302, 1303, 1304, 1306, 1313, 1325, 1326, 1329, 1332, 1336, 1340, 1341, 1344, 1355, 1358, 1362, 1363, 1364, 1379, 1381, 1383, 1384, 1386, 1387, 1389, 1393, 1395, 1396, 1397, 1398, 1402, 1404, 1406, 1422, 1423, 1424, 1425, 1426, 1431, 1433, 1434, 1435, 1436, 1438, 1446, 1447, 1450, 1457, 1462, 1463, 1465, 1467, 1475, 1479, 1481, 1483, 1486, 1487, 1488, 1489, 1497, 1498
48. **nerv. syst.** nervous system, other than CNS
54, 87, 93, 102, 110, 126, 127, 130, 140, 167, 188, 189, 194, 212, 244, 259, 277, 284, 313, 341, 361, 393, 415, 416, 431, 432, 441, 454, 459, 478, 481, 482, 492, 493, 494, 495, 496, 510, 514, 533, 560, 588, 595, 606, 638, 663, 737, 758, 760, 761, 762, 763, 764, 765, 766, 813, 851, 852, 854, 856, 865, 928, 936, 937, 968, 978, 1010, 1037, 1086, 1137, 1138, 1157, 1160, 1170, 1171, 1176, 1180, 1181, 1194, 1219, 1239, 1346, 1354, 1361, 1385, 1414, 1417, 1419, 1423, 1494
49. **respir.** respiration, including breath
1, 2, 3, 4, 12, 26, 30, 35, 42, 47, 49, 54, 58, 82, 92, 98, 106, 107, 109, 122, 126, 140, 141,

- 162, 183, 184, 185, 196, 200, 204, 209, 221, 222, 223, 228, 232, 233, 236, 243, 244, 251, 253, 255, 259, 265, 272, 291, 298, 353, 359, 379, 381, 389, 390, 392, 414, 417, 431, 434, 440, 446, 450, 456, 462, 465, 470, 471, 476, 482, 489, 490, 523, 526, 532, 540, 543, 557, 559, 560, 575, 579, 582, 606, 614, 620, 621, 622, 627, 632, 636, 638, 647, 654, 674, 687, 688, 693, 711, 717, 718, 724, 728, 732, 733, 755, 766, 791, 798, 808, 814, 835, 839, 842, 845, 846, 851, 861, 862, 877, 895, 904, 907, 908, 909, 912, 914, 915, 920, 930, 943, 950, 953, 954, 958, 959, 977, 979, 981, 983, 988, 1016, 1020, 1023, 1029, 1030, 1034, 1038, 1059, 1085, 1092, 1094, 1098, 1115, 1124, 1127, 1132, 1136, 1137, 1138, 1149, 1157, 1158, 1159, 1160, 1172, 1184, 1187, 1245, 1246, 1263, 1267, 1273, 1299, 1300, 1324, 1346, 1357, 1368, 1369, 1375, 1385, 1387, 1395, 1399, 1407, 1418, 1423, 1440, 1452, 1459, 1460, 1462, 1464, 1465, 1467, 1483, 1492, 1495
50. **senses** senses and sensation
12, 18, 87, 144, 188, 193, 205, 213, 244, 251, 259, 277, 288, 289, 331, 363, 379, 393, 422, 431, 480, 481, 482, 537, 555, 565, 582, 590, 599, 601, 603, 604, 606, 610, 638, 640, 641, 654, 762, 798, 801, 828, 837, 841, 843, 849, 852, 859, 860, 874, 875, 876, 915, 953, 984, 1067, 1096, 1113, 1133, 1136, 1137, 1138, 1139, 1140, 1141, 1142, 1193, 1239, 1266, 1354, 1361, 1370, 1385, 1414, 1423, 1427
51. **skel., muscle, skin** skeleton, muscle, skin
4, 6, 35, 42, 54, 69, 73, 74, 88, 98, 104, 126, 140, 146, 165, 183, 189, 207, 209, 210, 212, 244, 245, 251, 255, 259, 292, 295, 307, 332, 378, 431, 442, 470, 489, 490, 510, 527, 555, 577, 595, 621, 622, 633, 636, 638, 648, 654, 670, 696, 717, 722, 737, 744, 773, 781, 782, 802, 803, 806, 823, 833, 850, 856, 898, 899, 901, 922, 953, 956, 957, 968, 976, 1010, 1043, 1057, 1060, 1061, 1072, 1086, 1103, 1113, 1136, 1137, 1138, 1139, 1151, 1152, 1160, 1170, 1171, 1180, 1181, 1192, 1231, 1238, 1240, 1241, 1243, 1264, 1280, 1281, 1284, 1346, 1370, 1385, 1393, 1415, 1423, 1451, 1459, 1460, 1467, 1480, 1482, 1493, 1497

COMPOUNDS INTERACTING WITH ETHANOL

52. **alcohols** alcohols, other than ethanol, such as methanol, propanol, etc.
2, 5, 9, 10, 11, 12, 13, 23, 24, 62, 76, 89, 90,

- 98, 111, 116, 125, 138, 144, 156, 172, 247, 251, 269, 358, 379, 418, 431, 479, 480, 481, 482, 512, 611, 649, 671, 672, 698, 700, 710, 755, 760, 776, 811, 864, 896, 936, 937, 1035, 1037, 1054, 1057, 1096, 1122, 1123, 1124, 1125, 1136, 1137, 1138, 1139, 1140, 1141, 1142, 1143, 1144, 1157, 1175, 1198, 1242, 1276, 1303, 1307, 1308, 1355, 1422, 1423, 1433, 1437, 1439, 1484, 1495
53. **amphetamines** amphetamines (dexamphetamine, methamphetamine, etc.) 54, 68, 72, 76, 87, 130, 180, 182, 220, 258, 280, 281, 282, 308, 309, 371, 372, 374, 375, 398, 404, 429, 430, 454, 495, 496, 498, 504, 513, 514, 515, 535, 539, 540, 562, 574, 592, 603, 609, 612, 629, 650, 674, 719, 724, 734, 760, 771, 809, 815, 816, 839, 865, 876, 983, 984, 1037, 1066, 1078, 1083, 1105, 1106, 1107, 1126, 1127, 1128, 1149, 1160, 1174, 1226, 1244, 1261, 1262, 1269, 1284, 1295, 1336, 1337, 1372, 1420, 1423, 1449, 1453, 1456, 1457, 1470, 1476
54. **analeptics** analeptics (bemegride, meclogenoxate, etc.) 294, 404, 574, 765, 1219, 1229, 1357, 1423
55. **analg., antipyret.** analgesics and antipyretics (APC, phenacetin, etc.) 15, 23, 24, 36, 37, 45, 58, 62, 72, 85, 91, 99, 100, 105, 106, 107, 114, 121, 123, 133, 139, 145, 158, 164, 165, 167, 175, 216, 267, 272, 291, 295, 301, 313, 324, 333, 345, 362, 368, 369, 375, 403, 404, 413, 421, 423, 425, 426, 429, 430, 451, 452, 453, 456, 493, 500, 501, 502, 520, 521, 523, 527, 540, 552, 553, 578, 579, 584, 599, 617, 619, 620, 631, 634, 654, 664, 668, 669, 673, 674, 680, 688, 748, 765, 772, 776, 783, 784, 785, 789, 799, 807, 839, 850, 858, 869, 907, 908, 910, 924, 925, 926, 930, 951, 956, 958, 959, 996, 1001, 1028, 1029, 1037, 1046, 1047, 1051, 1066, 1089, 1092, 1108, 1110, 1116, 1117, 1193, 1201, 1203, 1208, 1210, 1211, 1212, 1213, 1214, 1215, 1229, 1230, 1233, 1239, 1266, 1276, 1279, 1282, 1283, 1297, 1301, 1313, 1356, 1366, 1370, 1382, 1389, 1391, 1397, 1398, 1399, 1400, 1401, 1407, 1408, 1423, 1425, 1426, 1430, 1435, 1448, 1453, 1459, 1460, 1481, 1482, 1492, 1498
56. **anesthetics** anesthetics (butamin, ether, cocaine, etc.) 2, 4, 6, 9, 14, 15, 35, 58, 93, 101, 103, 113, 162, 167, 179, 221, 222, 243, 257, 264, 302, 313, 328, 342, 354, 438, 442, 453, 462, 464, 520, 521, 527, 538, 546, 552, 588, 591, 594, 595, 621, 653, 670, 696, 719, 736, 737, 758, 766, 780, 850, 852, 865, 892, 929, 948, 956, 983, 992, 997, 1010, 1011, 1060, 1061, 1063, 1071, 1072, 1076, 1086, 1087, 1091, 1129, 1133, 1135, 1150, 1154, 1161, 1179, 1187, 1193, 1215, 1216, 1217, 1218, 1264, 1286, 1307, 1308, 1342, 1351, 1371, 1378, 1406, 1413, 1415, 1423, 1446, 1479, 1492, 1498
57. **anticonvulsants** anticonvulsants (carbamazepine, phenytoin, etc.) 61, 247, 389, 417, 477, 595, 630, 655, 656, 664, 707, 709, 760, 832, 957, 1060, 1061, 1313, 1423, 1466, 1498
58. **antidepressants** antidepressants (desimipramine, prolintane, etc.) 206, 235, 245, 299, 306, 312, 377, 404, 422, 432, 531, 547, 553, 599, 680, 683, 745, 757, 762, 765, 792, 800, 810, 867, 878, 879, 881, 882, 885, 886, 887, 913, 969, 1037, 1111, 1190, 1196, 1247, 1273, 1320, 1321, 1342, 1343, 1390, 1401, 1406, 1423, 1438, 1492, 1496, 1498
59. **anti-infectants** anti-infectants (penicillin, cycloserine, etc.) 1, 20, 21, 26, 29, 30, 64, 65, 78, 96, 120, 121, 133, 157, 158, 185, 207, 255, 257, 272, 273, 310, 332, 349, 362, 366, 392, 409, 437, 440, 455, 465, 466, 470, 485, 486, 487, 500, 507, 530, 532, 542, 551, 558, 561, 567, 568, 635, 654, 683, 690, 693, 695, 711, 712, 737, 739, 740, 741, 742, 743, 770, 774, 783, 784, 785, 795, 820, 821, 838, 840, 842, 846, 849, 857, 890, 902, 914, 915, 920, 943, 977, 995, 1022, 1031, 1034, 1037, 1052, 1066, 1067, 1081, 1085, 1100, 1132, 1134, 1150, 1156, 1186, 1211, 1212, 1213, 1219, 1229, 1236, 1239, 1252, 1263, 1265, 1271, 1275, 1287, 1288, 1297, 1301, 1309, 1310, 1328, 1360, 1365, 1368, 1397, 1398, 1401, 1402, 1408, 1419, 1423, 1434, 1453, 1454, 1481, 1484, 1498
60. **antispasmodics** antispasmodics (ethyl acetate, papaverine, etc.) 277, 322, 404, 595, 816, 936, 937, 939, 1055, 1175, 1325, 1423, 1492, 1498
61. **autocoids** autocoids (antihistamine, clemizole, etc.) 23, 24, 43, 44, 70, 87, 246, 320, 403, 431, 495, 496, 604, 621, 664, 677, 807, 861, 869, 1076, 1206, 1239, 1283, 1297, 1306, 1366, 1405, 1408, 1429, 1441, 1486, 1487, 1498
62. **autonomic agents** autonomic agents (parasympatholytics, parasympathomimetics, sympatholytics, sympathomimetics) 53, 62, 70, 102, 153, 160, 194, 216, 234, 237,

- 238, 301, 322, 353, 356, 413, 417, 431, 538, 539, 553, 620, 644, 664, 674, 694, 773, 791, 816, 818, 820, 839, 891, 901, 907, 908, 909, 910, 930, 956, 958, 959, 968, 983, 1071, 1077, 1100, 1126, 1127, 1128, 1149, 1154, 1180, 1181, 1206, 1276, 1329, 1345, 1349, 1413, 1419, 1420, 1423, 1424, 1427, 1451, 1453, 1459, 1460, 1498
63. **barbiturates** barbiturates (barbital, secobarbital, etc.)
9, 14, 15, 23, 24, 25, 32, 40, 41, 46, 47, 48, 57, 58, 66, 77, 92, 93, 94, 105, 121, 133, 155, 167, 175, 185, 187, 190, 191, 192, 196, 197, 199, 211, 217, 218, 219, 221, 222, 223, 224, 225, 226, 227, 228, 229, 243, 245, 246, 249, 261, 270, 278, 283, 304, 314, 315, 316, 326, 328, 330, 336, 337, 338, 339, 340, 343, 344, 346, 347, 348, 351, 356, 358, 360, 362, 395, 398, 404, 406, 407, 408, 426, 432, 438, 443, 445, 451, 452, 453, 460, 467, 468, 476, 491, 505, 519, 520, 521, 522, 526, 527, 531, 534, 535, 538, 540, 561, 564, 570, 571, 581, 591, 593, 595, 597, 600, 617, 619, 628, 632, 633, 641, 645, 646, 661, 662, 674, 677, 680, 684, 688, 692, 693, 702, 706, 707, 715, 717, 718, 719, 725, 756, 757, 759, 761, 762, 765, 783, 784, 785, 787, 792, 804, 807, 814, 818, 823, 825, 826, 827, 835, 839, 855, 861, 862, 863, 870, 876, 877, 887, 906, 907, 908, 909, 910, 916, 923, 924, 925, 926, 929, 961, 965, 970, 976, 983, 985, 989, 999, 1003, 1025, 1026, 1027, 1028, 1034, 1036, 1037, 1044, 1046, 1047, 1052, 1066, 1067, 1068, 1070, 1073, 1075, 1076, 1079, 1087, 1088, 1090, 1092, 1093, 1100, 1101, 1102, 1103, 1108, 1110, 1111, 1116, 1117, 1118, 1130, 1154, 1163, 1164, 1166, 1167, 1168, 1184, 1188, 1202, 1211, 1215, 1216, 1217, 1225, 1227, 1230, 1231, 1237, 1239, 1253, 1254, 1255, 1258, 1261, 1262, 1269, 1274, 1278, 1280, 1281, 1282, 1286, 1291, 1298, 1301, 1313, 1323, 1330, 1338, 1349, 1354, 1356, 1358, 1362, 1370, 1374, 1375, 1380, 1386, 1392, 1397, 1398, 1404, 1406, 1410, 1411, 1412, 1423, 1429, 1430, 1440, 1441, 1443, 1444, 1446, 1448, 1453, 1455, 1459, 1462, 1463, 1464, 1465, 1469, 1472, 1481, 1492, 1493, 1498
64. **coagulants** blood formation and coagulation agents (oxalic acid, phenprocoumon, etc.)
39, 61, 403, 431, 655, 656, 769, 816, 1037, 1066, 1287, 1432
65. **cardiovasc. agents** cardiovascular agents (digitalis, dopamine, etc.)
133, 247, 265, 353, 435, 523, 527, 719, 721, 760, 784, 785, 788, 791, 802, 803, 891, 975, 1030, 1037, 1066, 1100, 1149, 1180, 1293, 1358, 1364, 1405, 1421, 1423, 1444, 1498
66. **diagnost. agents** diagnostic agents (sodium benzoate, sodium iodide, etc.)
254, 620, 643, 648, 1063, 1180, 1240, 1260
67. **elect., water-bal. agents** electrolyte and water-balance agents (diuretics, ions, glucose, etc.)
95, 122, 153, 160, 165, 185, 193, 251, 257, 276, 323, 342, 353, 362, 369, 375, 379, 477, 500, 523, 542, 582, 618, 691, 695, 701, 718, 719, 734, 752, 769, 775, 807, 816, 844, 865, 877, 892, 906, 930, 960, 1014, 1015, 1064, 1088, 1100, 1123, 1142, 1145, 1170, 1206, 1219, 1242, 1287, 1288, 1297, 1325, 1358, 1359, 1365, 1366, 1383, 1393, 1423, 1449, 1451, 1459, 1460, 1481
68. **enzymes** enzymes (monoamine oxidase inhibitors, iproniazid, etc.)
33, 115, 133, 134, 268, 296, 349, 432, 643, 644, 814, 1034, 1099, 1100, 1166, 1272, 1273, 1396, 1400, 1424, 1435
69. **gastrointest. agents** gastrointestinal agents (meclozine, glutamic acid, etc.)
87, 95, 159, 163, 247, 295, 431, 439, 451, 495, 496, 542, 596, 613, 670, 760, 764, 785, 816, 819, 826, 827, 847, 849, 994, 1288, 1333, 1334, 1400, 1498
70. **hallucinogens** hallucinogens (cannabis, lysergide, etc.)
54, 63, 93, 177, 321, 351, 355, 436, 450, 498, 513, 514, 586, 636, 675, 694, 762, 836, 837, 841, 849, 850, 880, 884, 985, 1190, 1197, 1268, 1330, 1404, 1423, 1429
71. **hormones, hormone antag.** hormones and hormone antagonists (ACTH, insulin, etc.)
17, 22, 30, 42, 61, 105, 121, 128, 132, 133, 143, 146, 158, 160, 178, 198, 200, 208, 209, 210, 216, 231, 234, 243, 257, 286, 342, 375, 403, 414, 417, 420, 429, 430, 457, 477, 498, 499, 500, 513, 535, 557, 580, 643, 647, 655, 656, 657, 666, 674, 695, 719, 734, 744, 746, 749, 775, 796, 807, 824, 865, 892, 924, 925, 960, 962, 966, 979, 996, 1000, 1037, 1065, 1100, 1104, 1157, 1158, 1230, 1235, 1248, 1283, 1297, 1298, 1358, 1364, 1365, 1367, 1406, 1445, 1453, 1459, 1460, 1498
72. **indust. intox.** industrial intoxicants (carbon monoxide, gasoline, etc.)
119, 144, 149, 174, 232, 298, 303, 305, 373, 375, 390, 500, 537, 557, 626, 829, 897, 912, 953, 954, 960, 1050, 1053, 1054, 1069, 1131,

- 1147, 1185, 1221, 1243, 1335, 1395, 1408, 1450, 1467, 1498
73. **integ. syst. agents** integumentary system agents (camphor, menthol, etc.)
193, 1204, 1251, 1479
74. **miscellaneous** compounds used in manufacturing or not otherwise specified (alanine, mercury, etc.)
16, 60, 78, 90, 118, 124, 142, 169, 199, 241, 250, 254, 281, 334, 335, 349, 354, 384, 391, 410, 446, 459, 469, 500, 543, 546, 555, 563, 606, 607, 622, 624, 689, 695, 703, 716, 719, 737, 769, 816, 817, 839, 890, 897, 936, 937, 988, 991, 1037, 1063, 1089, 1104, 1115, 1131, 1167, 1260, 1271, 1277, 1293, 1305, 1307, 1308, 1313, 1346, 1358, 1385, 1401, 1414, 1467, 1477, 1498
75. **musculoskel. agents** musculoskeletal agents (antirheumatics, muscle relaxants, etc.)
82, 102, 185, 194, 561, 701, 816, 818, 839, 1037, 1100, 1154, 1170, 1292, 1319, 1357, 1419, 1444, 1498
76. **neoplast. agents** neoplastic agents (alloxan, urethane, etc.)
216, 300, 524, 542, 543, 825, 1087, 1092, 1431
77. **nutritive agents** nutritive agents (vitamins, nicotinamide, etc.)
30, 79, 136, 137, 139, 185, 205, 375, 376, 542, 583, 584, 618, 620, 688, 723, 750, 760, 769, 775, 816, 843, 886, 925, 933, 966, 1079, 1123, 1232, 1297, 1337, 1358, 1384, 1406, 1469, 1476, 1492
78. **respir. agents** respiratory agents (potassium iodide, tyloxapol, etc.)
435, 895, 1419
79. **sed., hypnot.** sedatives and hypnotics, (excluding barbiturates), such as apronal glutethimide, etc.
7, 8, 9, 30, 49, 50, 55, 69, 72, 75, 80, 83, 87, 104, 108, 112, 127, 133, 135, 145, 155, 161, 175, 188, 203, 204, 261, 264, 278, 279, 283, 301, 306, 321, 325, 328, 329, 344, 347, 349, 351, 356, 357, 358, 359, 364, 365, 375, 388, 389, 398, 403, 404, 411, 416, 436, 444, 456, 460, 472, 473, 474, 475, 488, 495, 496, 498, 500, 506, 523, 527, 540, 569, 585, 595, 619, 628, 631, 651, 652, 665, 673, 678, 680, 683, 684, 685, 686, 688, 691, 693, 707, 709, 717, 718, 731, 747, 757, 759, 761, 762, 763, 764, 765, 766, 783, 784, 785, 807, 816, 818, 820, 825, 826, 827, 832, 848, 858, 862, 863, 869, 877, 887, 913, 925, 985, 994, 1009, 1016, 1020, 1021, 1025, 1029, 1033, 1046, 1047, 1048, 1049, 1050, 1051, 1052, 1057, 1063, 1068, 1075, 1076, 1087, 1090, 1091, 1092, 1100, 1111, 1128, 1154, 1162, 1164, 1169, 1188, 1205, 1206, 1216, 1217, 1229, 1231, 1259, 1270, 1277, 1283, 1284, 1288, 1289, 1290, 1297, 1308, 1311, 1313, 1322, 1327, 1345, 1353, 1363, 1366, 1376, 1400, 1401, 1402, 1403, 1404, 1406, 1408, 1409, 1411, 1413, 1423, 1428, 1429, 1442, 1448, 1463, 1466, 1471, 1478, 1483, 1486, 1492, 1496, 1498
80. **stimulants** stimulants, excluding amphetamines (pemoline, caffeine, etc.)
15, 27, 28, 30, 31, 53, 54, 59, 73, 74, 76, 85, 117, 140, 154, 159, 167, 173, 213, 216, 240, 256, 271, 274, 276, 294, 322, 323, 349, 352, 353, 368, 370, 374, 375, 376, 404, 415, 422, 425, 428, 429, 430, 431, 432, 434, 436, 458, 460, 461, 471, 483, 484, 489, 490, 498, 499, 504, 513, 514, 520, 521, 528, 539, 542, 543, 545, 548, 549, 550, 559, 560, 564, 566, 569, 574, 577, 582, 592, 595, 598, 613, 615, 616, 620, 627, 629, 664, 674, 680, 684, 713, 719, 722, 724, 728, 730, 734, 746, 782, 791, 807, 808, 812, 816, 839, 845, 849, 853, 859, 860, 861, 865, 868, 893, 918, 922, 924, 928, 942, 950, 957, 964, 966, 983, 984, 985, 986, 989, 996, 1000, 1008, 1019, 1023, 1034, 1037, 1038, 1041, 1045, 1058, 1059, 1063, 1070, 1076, 1078, 1084, 1094, 1120, 1134, 1135, 1148, 1153, 1159, 1172, 1174, 1182, 1183, 1190, 1191, 1195, 1197, 1204, 1222, 1230, 1232, 1245, 1246, 1267, 1283, 1287, 1294, 1296, 1298, 1301, 1313, 1331, 1336, 1357, 1373, 1377, 1378, 1381, 1400, 1401, 1404, 1405, 1413, 1423, 1429, 1445, 1448, 1453, 1456, 1457, 1473, 1480, 1484, 1485, 1489, 1498
81. **tranquilizers** tranquilizers (chlordiazepoxide, reserpine, etc.)
19, 30, 38, 51, 53, 56, 68, 82, 93, 105, 115, 126, 127, 133, 152, 161, 166, 167, 168, 170, 176, 177, 178, 185, 187, 188, 189, 195, 214, 235, 239, 245, 246, 248, 249, 275, 283, 284, 285, 292, 297, 306, 307, 319, 327, 330, 338, 339, 340, 341, 344, 347, 350, 351, 357, 359, 363, 364, 365, 371, 373, 374, 385, 393, 394, 396, 402, 404, 416, 421, 423, 424, 426, 427, 429, 430, 432, 441, 448, 449, 452, 453, 460, 467, 468, 491, 492, 493, 494, 495, 496, 503, 506, 516, 517, 518, 520, 521, 527, 531, 535, 547, 552, 553, 554, 561, 569, 572, 573, 582, 587, 591, 592, 593, 597, 600, 601, 602, 605, 619, 621, 628, 631, 640, 642, 648, 663, 664, 676, 678, 679, 680, 681, 682, 683, 685, 686, 691, 708, 714, 715, 718, 719, 725, 732, 733,

754, 757, 761, 762, 763, 765, 773, 792, 797,
801, 804, 810, 822, 834, 839, 843, 858, 869,
871, 872, 873, 874, 875, 880, 883, 885, 887,
888, 898, 899, 900, 903, 904, 905, 913, 923,
926, 927, 929, 931, 932, 933, 944, 945, 946,
947, 971, 985, 987, 989, 990, 1002, 1007,
1024, 1034, 1037, 1039, 1042, 1062, 1066,
1067, 1068, 1070, 1076, 1087, 1089, 1100,
1109, 1111, 1112, 1113, 1114, 1155, 1160,
1161, 1166, 1169, 1184, 1191, 1194, 1199,
1203, 1207, 1219, 1223, 1228, 1229, 1230,
1258, 1261, 1262, 1273, 1280, 1281, 1285,
1298, 1301, 1313, 1320, 1321, 1322, 1326,
1349, 1350, 1352, 1357, 1366, 1369, 1375,
1376, 1394, 1400, 1405, 1406, 1408, 1423,
1427, 1428, 1429, 1430, 1441, 1454, 1473,
1481, 1496, 1498, 1499, 1500

82. unclass. ther. agents unclassified therapeutic agents (disulfiram, mercury iodide, etc.)

16, 30, 105, 121, 133, 142, 171, 199, 202, 207,
209, 234, 236, 242, 243, 247, 248, 253, 259,
262, 281, 284, 311, 319, 341, 349, 397, 399,
401, 412, 431, 446, 447, 500, 544, 626, 638,
643, 695, 720, 751, 760, 761, 762, 763, 764,
765, 769, 788, 790, 810, 813, 816, 960, 962,
963, 966, 1011, 1033, 1034, 1052, 1057, 1063,
1064, 1067, 1082, 1095, 1100, 1109, 1118,
1154, 1156, 1157, 1159, 1206, 1238, 1249,
1250, 1256, 1257, 1287, 1288, 1293, 1299,
1300, 1307, 1366, 1385, 1391, 1397, 1398,
1408, 1423, 1447, 1468, 1475, 1497, 1498

Author Index

- Aaker, H. 311
 Abbott, G. A. 1
 Abel, J. J. 2, 3
 Abreu, B. E. 4
 Abshagen, U. 5
 Accardi, V. 18
 Adams, F. J. 6
 Adams, J. E. 1326
 Adams, W. L. 7, 8
 Adefuin, J. 272, 273
 Adriani, J. 9
 Agner, K. 10, 11, 12, 13
 Ahlquist, R. P. 14, 15, 316
 Akabane, J. 16, 17
 Alajmo, B. 18
 Albert, J. R. 797
 Albert, S. N. 19, 394
 Aldighieri, J. 1192
 Alexander, C. S. 311
 Alha, A. 20, 21, 22, 23, 24, 358, 359, 739
 Allbritten, F. F. 476
 Allegri, A. 25
 Allen, E. V. 1470
 Allen, L. E. 797
 Allison, B. R. 26
 Allmark, M. G. 506
 Alman, R. W. 394
 Alstott, R. L. 27, 28
 Alvares, A. 1167
 Alvariñas, C. 744
 Alves, M. A. M. 29
 Amado Ledo, E. 30
 Amagat 31
 American Medical Association, Committee on
 Alcoholism and Addiction 32, 33
 Ammon, H. P. T. 34, 384
 Amorosi, O. 35
 Amsel, L. P. 36
 Andersen, A. H. 37
 Andersén, K. 38
 Anderson, R. C. 867
 Andoh, N. 998
 Anonymous 39, 40, 41, 42, 43, 44, 45, 46, 47,
 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59,
 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71,
 72, 73, 74, 75
 Aqvist, S. 161
 Arbuzov, S. Ia. 76
 Archer, J. D. 77
 Arima, Y. 78
 Ariyama, H. 79
 Ariyoshi, T. 80, 81
 Armour, P. S. 82
 Arnaud, G. 1192
 Arnold, W. 83
 Aron, T. 84
 Arullani, C. 85
 Arvola, A. 86
 Aschan, G. 87
 Aschkenasy-Lelu, P. 88
 Ashley, L. G. 1077
 Asser, E. 89, 90
 Astley, C. E. 91
 Aston, R. 92, 93, 94
 Aub, J. C. 120
 Aubinière 95
 Aufdermaur, M. 96
 Aull, J. C., Jr. 973
 Bacchin, P. 1165
 Backhouse, C. I. 97
 Baer, G. 98
 Bagdon, R. E. 1496
 Bagley, S. K. 612
 Baglioni, S. 1233
 Bakalář, E. 841
 Ballatore, C. 99, 100
 Balodis, K. 101, 102, 103
 Baraibar, E. 383
 Barbee, T. 280, 281
 Barbillion 104
 Barboriak, J. J. 105, 1474, 1475
 Bardisa, L. 913
 Bardoděj, Z. 106, 107, 108
 Barkman, R. 109
 Barlow, G. 702
 Barlow, O. W. 110
 Barry, H. 1423
 Bartlett, G. R. 111
 Bartley, A. H. 112
 Bartoníček, V. 113, 114
 Bassan, P. 1386
 Baštecký, J. 115
 Bastrup, J. T. 116
 Bateman, K. 1261, 1262
 Bauch, G. 529
 Baumann, H. 117
 Bäumlér, J. 619
 Baxter, R. C. 118
 Beard, J. D. 702
 Beck, R. A. 498
 Beck, W. V. 119
 Beckman, W. W. 120

- Bedaux, F. C. 121
 Beer, C. T. 122
 Behr, A. 123
 Belfrage, K. E. 10
 Bellus, E. 1065
 Bénard, H. 399
 Benassi, G. 124
 Bendfeldt, E. 132
 Bennett, I. 836
 Bennett, I. L., Jr. 125
 Benson, G. D. 777
 Berger, E. H. 251
 Berger, H. 127
 Berger, H. J. 126
 Bergstedt, M. 87
 Beringer, A. 128
 Berli, R. R. 210
 Berman, L. B. 1205
 Berndt, H. 129
 Bernstein, M. E. 130, 131
 Bersohn, I. 675
 Berthaux, N. 1220
 Bertram, F. 132
 Besendorf, H. 1062
 Bessman, S. P. 948
 Bester, J. F. 133
 Bethune, H. C. 134
 Betlheim, S. 135
 Biasotti, A. A. 403
 Bidwell, C. D. 163
 Biehl, B. 136, 137
 Bills, C. E. 138
 Binswanger, H. 139
 Binz, C. 140, 141
 Biondi, C. 142
 Bisset, G. W. 143
 Bizzi, A. 923
 Bjerver, K. 144
 Björnström, F. 145
 Blackman, H. J. 258
 Blažević, D. 135
 Blöch, J. 146
 Blomstrand, R. 147, 148
 Blood, F. R. 149
 Bloomer, H. A. 1205
 Blum, K. 150, 151
 Blumenthal, M. 152
 Bobier, P. -M. 153
 Boeree, B. H. 154
 Bogan, J. 155
 Bogen, E. 156
 Böhm, E. 157
 Böhmer, K. 158
 Boismare, F. 1315
 Boissier, J. -R. 159
 Bonjour, J. P. 160
 Bonnicksen, R. 161
 Boothby, W. M. 162
 Borden, T. A. 163
 Bouchier, I. A. D. 164
 Bougeant, H. 274
 Bourne, H. B. 165
 Bourque, R. 1155
 Bourrinet, M. P. 166
 Bourrinet, P. 167, 168, 1086, 1087
 Boutigny, P. -H. 169
 Bovill, J. G. 621
 Bowes, H. A. 170
 Bowman, K. M. 1023
 Boyd, E. M. 171
 Boyd, P. R. 1414
 Bradford, L. W. 403
 Brambilla, G. 1193
 Branch, A. 172
 Brandino, G. 173
 Braun, H. 174
 Bredenkamp, J. 1428
 Bressler, R. 1064
 Breyer, J. 1210
 Brighton, J. R. 1372
 Briglia, R. J. 175
 Brines, O. A. 251
 Brinner, L. 919
 Brodanová, M. 583
 Brodie, B. B. 176, 177, 1320, 1321
 Brohult, J. 178
 Broitman, S. A. 1260
 Broser, F. 179
 Brown, D. J. 27, 180
 Brown, E. A. 181
 Brown, H. 643
 Bruns, O. 182
 Brunton, L. 183
 Bruschi, C. A. 184
 Bruyneel, N. 233
 Brys, J. 1434
 Brzeski, Z. 185
 Büch, O. 1343
 Buller, F. 1484
 Bunn, L. 186
 Burbridge, T. N. 187, 678, 679, 1325, 1326, 1349
 Burger, E. 188, 189
 Buris, L. 190, 191, 192
 Burkitt, R. J. 193
 Burnam, W. 194
 Burner, M. 195
 Burrell, R. H. 134
 Burrows, E. H. 196

- Buttle, G. A. H. 197
 Büttner, H. 198, 199, 200, 201
 Butzengeiger, K. H. 202
 Byron, W. 1438
 Cabana, B. E. 203, 472, 473, 474, 475
 Cabarro, A. 744
 Cade, J. F. J. 204
 Caffi, M. 205
 Cahn, B. 754
 Cain, J. 282
 Caird, W. K. 206
 Cajgfinger, H. 312
 Calesnick, E. 207
 Cameron, D. F. 1337, 1476
 Campana, C. 208
 Camponovo, L. E. 236
 Canary, J. J. 718
 Canessa, I. 209
 Capron, M. J. 251
 Cardonnet, L. J. 210
 Carlton, P. L. 211
 Carpenter, C. P. 1275
 Carpenter, J. A. 212, 213, 214, 510
 Carpenter, R. K. 215
 Carpenter, T. M. 216
 Carratalá, R. 217, 218, 219, 220, 221, 222
 Carrière, G. 223, 224, 225, 226, 227, 228, 229
 Carroll, R. B. 230, 937, 938, 939
 Carroll, R. P. 541
 Carter, E. A. 706, 707
 Carulli, N. 231
 Cary, F. H. 125
 Cashaw, J. 643
 Casier, H. 232, 233, 234, 235, 292, 305
 Cassidy, P. 1438
 Castel-Branco, N. 795, 796
 Castelli, P. 1192
 Castex, M. R. 236
 Cataland, S. 649
 Cavalieri, U. 237, 238
 Cavanagh, R. C. 1260
 Cefalu, S. J. 1146
 Cembrano, J. 838
 Cerrato, C. M. 184
 Certhoux, J. 239
 Chantourelle 240
 Chapheau, M. 241, 242, 243
 Chapin, E. W. 1022
 Chapman, L. F. 244
 Chappell, A. G. 245
 Charest, M. -P. 573
 Chatagnon, C. 246
 Chatagnon, M. P. -A. 246
 Chauchard, P. 247, 248, 760, 761, 762, 763, 764, 765
 Chavez, R. 1235
 Chelton, L. G. 249
 Chevalier, A. 250
 Chew, W. B. 251
 Chiesara, E. 662
 Chiffot, M. J. 252
 Child, G. P. 253
 Chilian, O. 254
 Cho, M. H. 766
 Chomety, F. 160
 Christenson, P. J. 255
 Chubb, N. C. 612
 Cicchitti, F. 744
 Claeyss 256
 Clark, B. B. 257
 Clark, W. C. 258
 Cléménçon, H. 259
 Clements, E. L. 1442
 Clemm, W. N. 260
 Coddington, F. L. 846
 Coen, G. 1450
 Cohen, G. 1101, 1102
 Cohen, L. H. 540, 542
 Coldwell, B. B. 261, 262, 263, 264, 469, 1362, 1363, 1463, 1464, 1465
 Cole, L. J. 265
 Cole, V. V. 607
 Coleman, J. E. 1395
 Colon, P. L. 1260
 Comporti, M. 266
 Cook, W. A. 1444
 Cooke, A. R. 267, 501
 Coon, J. M. 1359
 Cooper, A. J. 268
 Cooper, J. R. 269
 Cooper, M. N. 125
 Copeman, P. R. v. d. R. 270
 Corkill, N. L. 271
 Cornish, H. H. 272, 273
 Cossa, P. 274
 Courtial, J. 399
 Courvoisier, S. 275
 Coutinho, E. M. 276
 Craig, R. D. 277
 Cramer, E. 278
 Cramer, F. 596
 Cramer, N. C. 283
 Creaven, P. J. 279, 280, 281, 1130, 1131, 1329
 Crémieux, A. 282
 Cruz, I. A. 283
 Cullumbine, H. 92, 93
 Culpan, R. H. 134

- Cummins, J. F. 284
 Custer, R. P. 1069
 Cutler, J. T. 890
 Cutting, W. C. 979, 980
 Czerwenka-Wenkstetten, H. 285
 Czyzyk, A. 286
 Dąbski, H. 1353
 D'Agostino, A. 873
 Dajani, R. M. 287
 Damrau, F. 288, 289, 290
 Dandiya, P. C. 291
 Danechmand, L. 235, 292
 Danger, W. 293
 Daugherty, M. 294
 Davalos, R. C. 744
 DaVanzo, J. P. 294
 Dave, K. C. 1181
 Davenport, H. W. 295
 Davidoff, E. 1105, 1106
 Davie, G. 1075
 Davies, E. B. 296
 Davis, D. A. 297
 Davis, J. H. 298
 Davis, M. E. 812
 Davis, V. E. 299
 Dawbarn, M. 300
 Dawson, W. S. 301
 De Blasi, S. 302
 De Schaepdryver, A. 235, 292
 Debray, C. 303
 Debry, G. 1156, 1158
 DeCarli, L. M. 787, 788, 789
 Degerli, I. U. 304, 1443, 1444
 Delaunois, A. L. 232, 305
 Delay, J. 306
 Delbue, C. 744
 Delga, J. 631
 Deluy, M. 1192
 Denefield, B. A. 1077
 Dengler, K. 307
 Deniker, P. 306
 Dent, J. Y. 308
 Dermer, L. 399
 Dérobert 309
 Dérobert, L. 310, 391
 Dérot, M. 310
 Derr, R. F. 311
 Dervillée 1133
 Desanti, E. 1192
 Descaux, J. 1071
 Despieres, G. 312
 Deutsch, H. 313
 Devenyi, P. 314, 315
 Di Luzio, N. R. 266, 317, 318, 921
 Dille, J. M. 15, 316, 1225
 Dimberg, R. 161, 319
 Ditman, K. S. 320, 321
 Divnogorskaia, N. N. 322
 Dixon, R. L. 323
 Dobbing, J. 324
 Dobkin, A. B. 766
 Dobronravov, P. A. 325
 Dobson, H. L. 1222
 Doenicke, A. 326, 327, 328, 329, 330, 445
 Doldt, H. 331
 Dolger, H. 332
 Dominiczak, K. 1186
 Dorset, V. J. 1173
 Douglas, D. 862, 863
 Dowd, P. J. 1176
 Dragomir, L. 1370
 Drouet, J. 737
 Druyan, R. 1395
 Dubitscher, F. 333
 Dubois, R. 334
 Ducrot, R. 275
 Duda, M. 450
 Düker, H. 335
 Dumont, E. 336
 Duncan, G. M. 419
 Dundee, J. W. 337, 338, 339, 340, 621
 Dupeyron, T. 310
 Duritz, G. 341, 1364
 Dürr, W. 1218
 Dürrigl, V. 640
 Dussel, E. 744
 Duverney, C. A. 19
 Dyke, L. H., Jr. 846
 Dyntarová, H. 1394
 Eade, N. R. 1396
 Eberhardt, D. R. 210
 Eckart, D. E. 342
 Edgecombe, P. C. E. 641
 Editorial 343, 344, 345, 346, 347, 348, 349,
 350, 351, 352, 353
 Edmondson, H. A. 354
 Eerola, R. 355, 356, 357, 358, 359, 1382
 Eggleton, M. G. 360, 361
 Ehring, F. 362
 Eicke, W. -J. 363
 Eickstedt, K. -W. von 364, 365
 Eirich, R. 366
 Eis, G. 367
 Ekberg, R. 538
 Elbel, H. 368, 369, 370, 371, 372, 373, 374,
 375, 376, 377
 Ellis, F. W. 1439
 Ellis, M. E. 265

- Elzay, R. P. 378
 Emerson, G. A. 4
 Engelhardt, K. 201
 Erickson, C. K. 194
 Erlanson, P. 379
 Erley, D. S. 1310
 Estable, J. J. 380, 381, 382, 383
 Estler, C. -J. 384
 Ettlinger, R. 385
 Etzler, K. 386, 387, 388, 389, 830
 Evans, R. L. 1273
 Ewing, P. L. 390
 Eyring, H. 635
 Fabre, R. 391
 Fahlgren, H. 385
 Farkas, I. S. 480, 481
 Farrier, R. M. 392
 Fasel, M. 451
 Fazekas, J. F. 19, 257, 393, 394, 581, 1207
 Fearn, H. J. 197, 395
 Federlin, C. 528
 Feldmann, H. 396
 Felzenberg, F. 776
 Ferguson, J. K. W. 397
 Ferneau, E. W., Jr. 967
 Ferrer Zanchi, A. G. 398
 Fiessinger, N. 399
 Figueroa, R. B. 400
 Filho, J. A. 276
 Filomusi-Guelfi, G. 401
 Finer, M. J. 402
 Fingl, E. 957
 Finkle, B. S. 403, 404
 Finnegan, J. K. 537
 Firmat, J. 236
 Fischer, E. 405
 Fischer, H. -D. 406, 407, 408, 409
 Fischer, I. 410
 Fisher, R. S. 411
 Fisher, W. 1395
 Fisk, A. J. 298
 Fiske, H. M. 412
 Fiske, J. P. 413
 FitzGerald, M. G. 414
 Flamm, S. 415
 Fleming, P. 416
 Fleming, R. 417
 Foerster, R. 418
 Forbes, J. C. 419
 Forgy, E. 321
 Forney, R. B. 27, 28, 130, 131, 180, 420, 421, 422, 423, 424, 425, 426, 427, 428, 429, 430, 431, 432, 598, 599, 600, 601, 602, 603, 604, 605, 608, 650, 651, 652, 653, 676, 836, 837
 Forsander, O. A. 433
 Förster, A. 434
 Forster, F. C. 435
 Forster, F. M. 1309
 Fort, J. 436
 Fournel, J. 275
 Foxell, A. W. H. 437
 Frahm, M. 438, 931
 Franc, J. 1249, 1250
 Francescato, F. 1492
 Francis, C. R. 439
 Franco, S. 440
 Frankenhaeuser, M. 441
 Fraser, D. B. 442
 Fraser, H. F. 443
 Freeman, J. 444
 Freinkel, N. 1189
 Frey, H. -H. 445, 826
 Fridman, V. 446
 Friedländer, A. 447
 Friedman, S. L. 448
 Friend, D. G. 284
 Fritz, H. 379
 Fröberg, J. 441, 449
 Frommel, E. 450, 451, 452, 453
 Fuchs, G. 454
 Fühner, H. 455, 456
 Fuhrman, F. A. 457
 Fuhrmann, J. 136, 137
 Funk, W. 321
 Gabriel, C. L. 458
 Gaddie, R. 414
 Gaede, D. 459
 Gaisbauer, G. 460
 Gallard, T. 461
 Galloway, D. H. 462
 Gamble, N. J. 463
 Gang, H. 1165, 1166, 1168
 Ganz, V. 464
 Garat, J. 758, 759
 Gardner, G. H. 465, 740
 Garson, H. 896
 Gary Bobo, C. 631
 Gates, P. W. 601
 Gavitt, J. 1266
 Gay, A. 466
 Gay, R. 1192
 Gebhart, G. F. 467, 468
 Geller, I. 150, 151
 Genest, K. 262, 469
 Geraghty, F. J. 470
 Gerard, J. 737
 Gerard, R. W. 843
 Gershoff, S. N. 1260

- Gervais, D. M. 471
 Gessner, P. K. 203, 472, 473, 474, 475
 Gettler, D. T. 476
 Gey, K. F. 1062
 Ghandour-Mnaymneh, L. 287
 Ghosh, J. J. 477
 Gibson, R. D. 835
 Giese, E. 478
 Gilger, A. P. 479, 480, 481, 482
 Gillissen, G. 698, 699, 700
 Giorgi, A. P. 927
 Girard 483
 Girard, M. 310
 Girdlestone, T. M. 484
 Glaesel, H. U. 307
 Glass, F. 485, 486, 487, 488
 Gold, E. 1190
 Gold, H. 489, 490
 Gold-Aubert, P. 491
 Goldberg, L. 87, 144, 441, 492, 493, 494, 495, 496, 497, 572, 681, 682, 684
 Goldfarb, W. 1023
 Goldstein, L. 498, 513, 515
 Goodell, H. 1482
 Goodman, J. I. 1436, 1437
 Goreczky, L. 499
 Göres, E. 500
 Gossow, H. 485
 Goulston, K. 501
 Grabill, F. J. 502
 Grady, R. W. 503
 Graf, O. 504
 Graham, J. D. P. 505
 Graham, R. C. B. 506
 Gray, I. 507
 Gray, M. G. 916
 Greenberg, L. 508
 Greenberg, L. A. 509, 510, 511, 512, 540, 541, 542, 936, 937, 938, 939
 Greenberg, R. E. 513, 514
 Greenberg, R. S. 515
 Greenfield, A. R. 516, 517
 Greenhouse, H. R. 518
 Greiser, E. 519, 520, 521, 522
 Grezzi, J. W. 380, 381, 382, 383
 Grier, W. F. 523
 Grilichess, R. 524
 Grose, W. 645
 Grove, R. C. 465
 Groves, J. W. 525
 Gruber, C. M., Jr. 526
 Grugni, C. 527
 Grüner, O. 528, 529
 Grymiński, J. 530
 Guarneri, A. 858
 Gugler, R. 531
 Guild, W. R. 532
 Gumbel, B. 533
 Gunning, B. 1200
 Gupta, R. C. 534
 Gupta, R. S. 823
 Gus'kov, V. S. 535
 Gustafson, R. K. 465, 740
 Gutschmidt, J. 536
 Guy, J. 1315
 Haag, H. B. 537, 665, 954, 1090
 Hadengue 309
 Hadji-Dimo, A. A. 538
 Haffner, F. 539
 Haggard, H. W. 540, 541, 542
 Hagstam, K. -E. 379
 Hake, C. L. 1310
 Hald, J. 543, 544
 Hall, A. J. 545
 Hall, C. R. 1322, 1323
 Hall, E. M. 354
 Haller, E. 546
 Hallermann, W. 578
 Halliwell, G. 547
 Hamacher, J. 1498
 Hameau 548, 549
 Hamilton, R. 550
 Hammes, E. M., Jr. 551
 Händel, K. 552, 553, 554
 Handwerker, J. V., Jr. 555
 Hansman, F. S. 556
 Hardinge, M. G. 1053, 1054
 Hardy, J. D. 1482
 Harger, R. N. 557
 Harlan, J. 649
 Harris, F. H. 558
 Hartman, A. 266
 Hartmann, A. 559
 Haskins, H. D. 560
 Hatfield, G. K. 561
 Hauschild, F. 562, 563
 Hawkins, R. D. 644
 Hay, M. G. 973
 Hayashida, K. 1267
 Hayes, W. N. 659, 660
 Haynes, H. 1395
 Hazleton, L. W. 564
 Hebbelinck, M. 292
 Heberlein, W. 1125
 Heck, K. 565
 Heidenreich, O. 566
 Heilner, E. 567
 Hein, J. 568

- Heinrichs, K. M. 569
 Hellems, H. K. 1103
 Hellerman, R. C. 564
 Hellström, R. 13
 Henley, K. S. 570
 Hensley, W. J. 118
 Herken, W. 1125
 Hermanns, A. 571
 Hermans, W. 235
 Hernández-Peón, R. 572
 Herr, F. 573, 1390
 Herxheimer, A. 1274
 Hess, J. 574
 Hesse, E. 575, 576
 Hessling, B. 577
 Heubner, W. 578
 Higgins, J. A. 579
 Hillbom, M. 580
 Himwich, H. E. 581
 Hine, C. H. 582, 1325, 1446, 1447
 Hockmuth, L. 502
 Hodges, J. R. 197, 395
 Hodnett, N. 1322, 1323
 Hoenig, V. 583
 Hoenle, R. 387, 389
 Hofacker, U. 584
 Hoffer, A. 585, 586, 587
 Hofmann, G. 285
 Holten, C. H. 588
 Höök, O. 11, 12
 Hopkins, H. H. 589
 Hopkins, W. K. 590
 Horáček, J. 654
 Horn, G. 591
 Horváth, D. 592
 Horvath, S. M. 1472
 Horvath, T. B. 1471
 Hoskovec, J. 593
 Höweler, A. 594
 Hubach, H. 595
 Hughes, D. T. D. 596
 Hughes, D. W. 262, 469
 Hughes, F. W. 130, 131, 180, 421, 422, 423, 424, 425, 426, 427, 428, 429, 431, 432, 597, 598, 599, 600, 601, 602, 603, 604, 605, 650, 651, 652, 676, 715
 Hugon, L. 606
 Hulpieu, H. R. 420, 421, 423, 557, 607, 608
 Hunsicker, H. 609
 Hunt, R. 610
 Hunter, J. M. 181
 Huriez, C. 223, 224, 225, 226, 227, 228, 229
 Hurst, H. 611
 Hurst, P. M. 612
 Husemann, T. 613
 Hutchens, J. O. 614
 Hutterer, F. 1162
 Iaroshevski, S. 615, 616
 Iber, F. L. 655, 656, 657, 658
 Idestrom, C. -M. 617
 Iida, S. 618
 Ikomi, F. 16
 Im Obersteg, J. 619, 681, 682, 684
 Imrie, J. A. 620
 Infante, R. 1220
 Ingalls, J. W. 508
 Ingalls, J. W., Jr. 448
 Ingle, D. 1177
 Inglis, J. 206
 Ingvar, D. H. 538
 Isaac, M. 337, 338, 339, 340, 621
 Isbell, H. 443
 Isokoski, M. 739
 Isotalo, A. 23, 24
 Israel, Y. 622, 1384
 Issekutz, B. von 623
 Isselbacher, K. J. 706, 707
 Ivy, A. C. 1153
 Iwai, J. 624, 625
 Jacobsen, E. 543, 544, 626
 Jäger, G. 445
 Jahn, E. 807
 Jaillet, J. 627
 Jain, N. C. 651, 652, 653
 James, I. P. 97
 Janiszewski, H. 628
 Janitzki, U. 1199
 Jansen, G. 629, 1174
 Janz, D. 630
 Jaulmes, C. 631
 Jensen, K. B. 805
 Jetter, W. W. 632, 633
 Jofre de Breyer, I. J. 634
 Johannsmeier, K. 1024
 Johansson, Ö. 1184
 Johnson, F. H. 635
 Johnson, L. V. 479
 Johnson, N. K. 443
 Johnston, W. W. 636
 Jolie, J. 233
 Jones, A. L. 1237
 Jones, R. T. 637
 Jordi, A. 638
 Joron, G. 862
 Joron, G. E. 863
 Joseph, A. D. 1180, 1181
 Josserand, M. 639
 Joswig, E. H. 386, 387, 389

- Jouany, J. M. 737
 Jovanovic, U. J. 640
 Joyce, C. R. B. 641
 Joye, E. 450
 Jusko, A. G. 941
 Juul, P. 642
 Kagan, J. R. 1023
 Kahil, M. E. 643
 Kakulas, B. A. 886
 Kalant, H. 644, 645, 646, 677
 Káldor, A. 647, 1065
 Kalsner, S. 648
 Kane, R. L. 649
 Kang, L. 294
 Kanyuck, D. O. 867
 Kaplan, H. L. 650, 651, 652, 653
 Karmin, M. 1266
 Karppanen, H. 1375, 1376
 Kass, W. A. 1474
 Kassil, G. 1305
 Kastor, O. 654
 Kater, R. M. H. 655, 656, 657, 658
 Katkin, E. S. 659, 660, 1339
 Kato, R. 661, 662, 663
 Kautzsch, E. 1314
 Kawahara, M. 664
 Kaye, S. 665
 Kaymakçalan, S. 666
 Kearns, W. 635
 Keeser, E. 667
 Keith, E. F. 1496
 Kellogg, J. H. 668, 669
 Kelly, E. L. 843
 Kelly, J. A. 670
 Kempinski, H. 713
 Kendal, L. P. 671, 672
 Kennard, D. A. 641
 Keokosky, W. Z. 776
 Kessler, A. 673
 Kettenmeyer, G. 674
 Kew, M. C. 675
 Khan, A. U. 676
 Khanna, J. M. 622, 646, 677
 Khouw, L. B. 678, 679
 Kibrick, E. 680
 Kielholz, P. 681, 682, 683, 684, 685, 686
 Kiese, M. 459
 Kiessling, K. -H. 687
 Kieve, R. 688
 Kinard, F. W. 973
 King, P. D. 1499, 1500
 Kini, M. M. 269
 Kinsell, L. W. 1200
 Kiplinger, G. F. 836, 837
 Kirchheim, D. 689
 Kisch, B. 1328
 Kissel, P. 1158
 Kitto, W. 690
 Kiyasu, W. 120
 Kjølstad, T. 691
 Klaasen, C. D. 692
 Klein, H. 693, 694, 695
 Klein, H. A. 696
 Kleinert, H. 329, 330
 Kliewe, H. 697, 698, 699, 700
 Klotz, A. P. 400
 Knick, B. 701
 Knight, G. J. 596
 Knott, D. H. 702
 Koe, B. K. 703, 704
 Koelsch, F. 705
 Koetschet, P. 275
 Koff, R. S. 706, 707
 Kofman, O. 708
 Kofoed, J. 534
 Kohei, H. 17
 Köhler, C. 387, 389, 709
 Koivusalo, M. 710
 Kolodny, A. L. 711
 Kolsky, M. 275
 Konwaler, B. E. 712
 Koopmann, H. 713
 Kopf, R. 714
 Kopmann, E. 715
 Koppányi, T. 716, 717, 718, 719
 Korablev, M. V. 720
 Kordač, V. 583
 Kordecki, R. 721
 Koroxenidis, G. 1103
 Korzis, J. 1491
 Kósa, F. 722
 Kotoku, S. 723
 Kovach, R. 1205
 Kraft, H. -G. 724
 Krantz, J. C., Jr. 725
 Kratzsch, E. 726
 Krivucová, M. 106
 Krop, S. 297, 1438
 Krug, W. 727
 Krull, G. 728
 Kryspin-Exner, K. 285
 Kubička, J. 729
 Kuczyński, L. 185
 Kudrin, A. N. 730
 Kuenssberg, E. V. 731
 Kugler, J. 328, 329
 Kulisiewicz, T. A. 732, 733, 734
 Kuntzman, R. 1167

- Kunz, H. 139
 Kunz, H. A. 1343
 Kup, G. 735
 Kutob, S. D. 736
 Kutschke, I. 129
 Laborit, H. 737
 Labourt, F. E. 236
 Lachnit, V. 738
 Lacroix, J. 491
 Laiho, K. 739
 Lamarche, M. 1156, 1157, 1158, 1159
 Lamson, P. D. 465, 740, 741, 742, 743
 Landabure, P. B. 744
 Landauer, A. A. 745, 888, 1039
 Láng, S. 746
 Lange, H. -J. 747
 Langman, M. J. S. 748
 Larsen, J. A. 749, 750, 824
 Larsen, V. 543, 544, 588
 Larson, P. S. 537
 Laties, V. G. 1449
 Läuppi, E. 751, 752, 753
 Lawton, M. P. 754
 Layne, E. C. 948
 Le Bourhis, B. 159
 Le Bourhis, J. 758
 Le Breton, E. 300
 Le Breton, R. 757, 758, 759
 Le Guiner, G. 391
 Leach, H. 811
 Leaf, G. 755
 Lebel, G. 1155
 LeBlanc, E. 756
 Lech, C. C. 1475
 Lechat, P. 1315
 Lecoq, R. 247, 248, 760, 761, 762, 763, 764, 765
 Ledebur, I. von 450, 452
 Lederer, H. 893
 Lee, P. K. Y. 766
 Lefevre, A. 894
 Lehan, P. H. 1103
 Lehman, A. J. 1439
 Lehman, E. G. 1022
 Lehmann, G. 1389
 Leiderman, P. H. 864
 Leithoff, H. 1490
 Leibach, W. K. 767, 768
 Leloir, L. F. 769
 LeMessurier, D. H. 845
 Lendle, L. 770
 Lenhardt, A. 146
 Lennart, L. 178
 Leonard, B. E. 771
 Leonards, J. R. 772
 Lerner, J. 773
 Lester, D. 509, 774, 775, 776, 777
 Leuschner, A. 778
 Leuschner, F. 778
 Leutner, V. 1429
 Levin, W. 1167
 Levy, G. 36, 779
 Lewis, D. J. 780
 Lewis, W. 781
 Lewis, W. B. 782
 Leyrie, J. 306
 Lickint, F. 783, 784, 785
 Liddy, E. 288, 289, 290
 Lieber, C. S. 786, 787, 788, 789, 790, 894, 1161, 1162, 1163, 1164, 1165, 1166, 1167, 1168
 Liebhardt, E. 329
 Lienert, G. A. 931, 932
 Liljeberg, J. A. 1254
 Liljenberg, B. 379
 Lin, R. 622
 Linck, K. 791
 Lind, N. 792
 Lindsly, H. 793
 Linke, H. 794
 Lisboa, P. E. 795, 796
 Lish, P. M. 797
 List, P. H. 798
 Lob, M. 799
 Löbkens, K. 438
 Lockett, M. F. 800
 Loitzl, E. 801
 Lokchina, E. 1305
 Lolli, G. 1234
 Loomis, H. P. 802, 803
 Loomis, T. A. 804, 1270
 Loredó, A. 744
 Lowe, F. H. 1119
 Lowenstein, L. M. 967
 Lu, F. C. 506, 999
 Lubash, G. D. 977
 Lucero, R. J. 1251
 Ludwig, O. 529
 Lui, S. 706, 707
 Lundquist, F. 805
 Lundsgaard, E. 806
 Lundt, P. V. 807
 Luton 808
 Lyczewska, J. 530
 Lynch, R. 321
 Macaud, G. 809
 MacCallum, W. A. G. 810
 Macht, D. I. 811, 812
 MacKenna, R. W. 813

- MacLean, A. R. 1470
 MacLeod, I. 814
 MacLeod, L. D. 215, 815, 816, 817, 818, 819, 820
 MacMahon, H. E. 821
 Madan, B. R. 822, 823
 Madsen, J. 749, 824
 Maehly, A. 161
 Maengwyn-Davies, G. D. 718, 719
 Mafart, Y. 857
 Magnus, R. V. 268
 Magnussen, M. P. 825, 826, 827
 Maharajh, M. 397
 Maire, E. D. 465, 740
 Maling, H. M. 181
 Malins, J. M. 414
 Mallach, H. J. 386, 387, 389, 485, 486, 487, 488, 565, 828, 829, 830, 831, 832, 833, 834, 854, 1040, 1198
 Mallory, T. B. 120
 Malone, M. H. 835
 Manenti, F. 231
 Manno, J. E. 836, 837
 Marchese, S. 911
 Mardones, J. 838, 839, 840
 Mareček, P. 841
 Markham, T. N. 842
 Marquardt, P. 1302
 Marquis, D. G. 843
 Marseille 844
 Marshall, E. K., Jr. 845, 1033
 Marshall, S. V. 556
 Marshman, J. 646
 Martin du Pan, R. 847
 Martin, E. 303
 Martin, H. 854
 Martin, W. B. 846
 Mathieu, P. 848
 Matsumura, R. 17
 Mattison, J. B. 849, 850
 Matussewitsch, I. S. 851
 Maughs, G. M. B. 852
 Mauriac 853
 Maxwell, W. B. 1363
 Mayer, B. 565
 Mayer, K. 565, 854
 Mayer, R. M. 855
 Mayrant, W. 856
 Mazaud, R. 857
 Mazoué, H. 247, 248, 760, 761, 762, 763, 764, 765
 Mazzucchelli, B. 858
 McArthur, C. 1372
 McAtee, O. B. 947, 1499, 1500
 McCabe, E. R. B. 948
 McCall, A. B. 949
 McCrea, F. D. 950
 McCrudden, F. H. 951
 McGee, C. J. 952
 McGuigan, H. A. 579
 McGuire, L. W. 953
 McIntire, R. 780
 McIsaac, W. M. 1329
 McKennis, H., Jr. 954
 McLean, A. E. M. 955
 McLean, A. J. 740
 McLean, R. 633
 McMahon, T. M. 614
 McMechan, F. H. 956
 McQuarrie, D. G. 957
 McRorie, N. 1190
 Meerhoff, A. 859, 860
 Meerhoff, W. 859, 860
 Meerlo, J. A. M. 861
 Meldolesi, J. 1386
 Mella, I. 209
 Melville, K. I. 862, 863
 Ménager, M. J. 310
 Mendelson, J. 864
 Menninger-Lerchenthal, E. 865
 Menozzi, C. 1194
 Mercure, J. 1155
 Merino, A. 913
 Merrill, J. P. 532
 Meyer, A. 866
 Meyering, E. 1389
 Meyers, D. B. 867
 Mezey, K. 868
 Michel, E. 869
 Mickat, K. 870
 Mihăilescu, M. 1000
 Mikkonen, H. 871, 872
 Miller, A. I. 873
 Miller, J. G. 843, 874, 875
 Miller, K. E. 779
 Miller, M. J. 1
 Miller, M. M. 876, 877
 Milner, G. 745, 800, 878, 879, 880, 881, 882, 883, 884, 885, 886, 887, 888, 1039
 Min, P. 889
 Minot, A. S. 743, 890
 Minsky, R. 873
 Minz, S. 891
 Mirone, L. 927
 Mirsky, I. A. 892, 893
 Misra, P. S. 894, 1166
 Mitchell, C. L. 467, 468
 Mitchell, G. L., Jr. 125
 Mitchell, S. W. 895

- Miya, T. S. 835
 Moeschlin, S. 896
 Mohnike, G. 286
 Mohr, L. 897
 Molenda, R. 898, 899, 900, 901, 902, 903, 904, 905
 Molinari, G. 906
 Møller, K. O. 907, 908, 909, 910
 Montale, P. 466, 911
 Montañés del Olmo, E. 912
 Montoya, G. 913
 Moon, H. D. 914, 915
 Mooney, H. B. 320
 Moore, M. 916
 Moragne, N. H. 917
 Morey, H. C. 918
 Morgan, A. F. 919
 Morgan, A. M. 1364
 Morgan, E. L. 920
 Morgan, J. C. 921
 Morin 922
 Morini, M. T. 1386
 Morpurgo, C. 1343
 Morrissey, R. W. 257
 Morselli, P. L. 923
 Morton, R. C. 9
 Moschos, C. B. 1103
 Moss, T. 321
 Mueller, B. 924, 925, 926
 Muheim, E. 96
 Muller, B. P. 927
 Müller-Limmroth, W. 928
 Müller-Plettenberg, D. 929
 Mundeleer, P. 930
 Munkelt, P. 931, 932
 Muñoz, E. 838
 Muñoz, J. M. 769
 Muraoka, H. 933
 Muratorio, J. 210
 Murphree, H. B. 934, 935, 936, 937, 938, 939, 940, 941
 Mutke, P. H. C. 942
 Myatt, A. V. 943
 Myers, R. D. 944, 1379
 Myers, R. O. 354
 Myerson, A. 1126, 1127, 1128
 Myrsten, A. -L. 441, 945, 946
 Naalsund, O. 958, 959
 Nadeau, G. 960
 Nagasawa, H. T. 311
 Nagy, J. 961
 Nakanishi, S. 17, 962, 963
 Nash, C. W. 1337, 1476
 Nash, H. 964
 Nasilowski, W. 965
 Nason, Z. M. 966
 Nathan, P. E. 967
 Neal, G. L. 1042
 Neely, E. A. 968
 Neidhardt, H. 969
 Nelemans, F. A. 970, 971, 972
 Nelson, G. H. 973, 974
 Nelson, N. 892
 Nessling, W. 699
 Neumann, W. 975
 Neumeyer, L. 976
 New, P. S. 977
 Newell, G. W. 978
 Newman, E. J. 984, 1273
 Newman, H. W. 979, 980, 981, 982, 983, 984, 1271, 1273
 Newman, W. H. 1374
 Nichols, J. L. 985
 Niedeggen, G. 986
 Nielsen, G. L. 987
 Nielsen Kudsk, F. 988
 Nieschulz, O. 989, 990
 Niggemeier, K. 991
 Nikki, P. 992
 Nilsson, C. G. 13
 Nishioka, T. 993
 Nizard, I. 757
 Nobes, P. 994
 Nostiz, H. 995
 Notz-Schwarz, I. von 996
 Novi, M. 997
 Noyes, C. B., Jr. 712
 Nukada, T. 998
 Nyiogi, S. K. 999
 Oardă, M. 1000
 Oberlandesgericht Celle [Superior District Appeal Court, Celle] 1001
 Oberlandesgericht Frankfurt [Superior District Appeal Court, Frankfurt] 1002
 Oberlandesgericht Hamburg [Superior District Appeal Court, Hamburg] 1003
 Oberlandesgericht Hamm [Superior District Appeal Court, Hamm] 1004, 1005, 1006, 1007
 Oberlandesgericht Oldenburg [Superior District Appeal Court, Oldenburg] 1008
 Obrzut, A. 898, 899, 901
 Oehme, P. 1009
 Oelkers, H. A. 1010, 1011
 Oelssner, W. 406, 407
 Ogata, H. 17
 Ogden, E. 1012
 Ogg, G. J. 134
 Oldewurtel, H. A. 1103

- Oliveras, E. J. 1013
 Olszyska, L. 1014, 1015, 1016, 1017, 1018, 1019, 1020, 1021
 Onyett, H. P. 608
 Orahovats, P. D. 1022
 Orenstein, L. L. 1023
 Orzel, R. A. 1452
 Osmond, H. 586
 Osterhaus, E. 1024, 1025, 1026, 1027, 1028, 1029
 O'Sullivan, D. J. 414
 Ott, I. 1030
 Otto, B. S. 1031
 Otto, H. 132
 Overholt, B. F. 1032
 Owens, A. H., Jr. 1033
 Paasonen, M. K. 1034
 Paganoni, C. 205
 Pandit, S. K. 621
 Panjwani, M. H. 1180
 Pantaleoni, M. 1035
 Papas, P. N. 184
 Paradise, R. R. 1036
 Parker, W. J. 1037
 Parkes, M. W. 792
 Parrish, A. E. 283
 Pascalis, B. 1038
 Patman, J. 745, 1039
 Paul, C. J. 1363
 Paulus, W. 1040, 1179
 Pawan, G. L. S. 1041
 Payan, H. 1192
 Pearce, J. 1368
 Pearson, R. G. 1042
 Pecora, L. J. 1043
 Pengsritong, K. 1044
 Peris, G. 466, 911
 Perisson, J. 1045
 Perman, E. S. 109
 Peršić, N. 135
 Pessoa, S. B. 1265
 Peter, H. 1046, 1047
 Peters, E. L. 797
 Peters, G. 160
 Peters, U. H. 1048, 1049, 1050, 1051, 1052
 Peterson, D. I. 1053, 1054
 Peterson, J. E. 1053, 1054
 Petit, D. 1220
 Petruch, F. 1218
 Pfeifer, E. 1055
 Pfeiffer, C. C. 513
 Phelip, H. 312
 Philip, G. E. 1056
 Phillips, R. D. 1496
 Philpott, N. W. 165
 Pickford, L. M. 1057
 Pietschmann, H. 738
 Piette, Y. 235
 Piker, P. 893, 1058
 Pikkarainen, P. 580
 Pilcher, J. D. 1059
 Pilot, M. L. 518
 Pilström, L. 687
 Pirkner, F. 1060, 1061
 Plaa, C. B. 919
 Plaa, G. L. 468, 736
 Platonow, N. 263
 Pletscher, A. 1062
 Plochmann, E. 1063
 Plummer, C. W. 411
 Podgainy, H. 1064
 Podolsky, B. 614
 Pogátsa, G. 647, 1065
 Pohl, W. 1066
 Pokorný, F. 106
 Pöldinger, W. 681, 682, 683, 684, 1067, 1068
 Pons, C. A. 1069
 Ponsold, A. 1070
 Porat, B. von 11, 12
 Portmann, G. 1071
 Portwich, F. 198, 201
 Potts, A. M. 479, 480, 481
 Potts, J. L. 1077
 Powell, S. D. 1072
 Prabhaker, V. 1098
 Prag, J. J. 1073, 1074
 Preller, A. C. N. 1075
 Preston, J. E. 258
 Pribilla, O. 1076
 Price, L. M. 934, 935, 936
 Proctor, C. D. 1077
 Prouty, R. W. 1364
 Pruitt, D. G. 660, 1339
 Prüll, G. 1078
 Puech, M. 274
 Pullar-Strecker, H. 1079, 1080, 1081, 1082
 Püllen, C. 1083
 Pusch, H. 1084
 Quadland, H. P. 1085
 Quadri, A. 237, 238
 Quastel, J. H. 122, 477
 Quevauviller, A. 1086, 1087
 Quinton, R. M. 547
 Rabattu, J. 282
 Rachek 1088
 Radlow, R. 612
 Rakieten, N. 540, 1089
 Rall, D. P. 323

- Ramanathan, A. N. 671, 672
 Ramet, M. 239
 Rampal, C. 1192
 Ramsey, H. 1090
 Ramseyer, A. 681, 682, 684
 Rana, P. K. 1181
 Randall, L. O. 1496
 Rankin, J. G. 1471
 Ransom, F. 1091
 Rapoport, A. 843
 Raspopova, T. V. 1092
 Ratcliffe, F. 1093
 Ratner, K. S. 1160
 Rauch, H. W. M. 1094
 Rauschke, J. 1095
 Ravina, A. 1096
 Raymond, A. F. 916
 Raynes, A. E. 1097
 Rea, E. 393
 Rea, E. L. 19, 1207
 Reddy, D. B. 1098
 Reddy, D. G. 1098
 Redetzki, H. M. 1099, 1100
 Redmond, G. 1101, 1102
 Reese, W. N. 1130
 Reese, W. N., Jr. 1131
 Regan, T. J. 1103
 Regoli, D. 160
 Reichard, H. 13, 178
 Reid, C. H., Jr. 1104
 Reifenstein, E. C., Jr. 1105, 1106, 1107
 Reilly, J. 1438
 Reinartz, E. F. K. 1108
 Reinert, R. E. 1109
 Reinhard, J. F. 1110
 Reinke, D. R. 978
 Reis, G. von 385
 Reisby, N. 1111, 1112, 1113, 1114
 Reith, H. 798
 Rejsek, K. 1115
 Remmer, H. 80, 1116, 1117
 Remy, E. 1497
 Rentschler, E. 1118
 Reuning, R. H. 779
 Reutt, H. 721
 Revol, L. 848
 Reynolds, D. 417
 Reynolds, W. A. 1119
 Rich, A. L. 503
 Richards, A. B. 130, 180, 605, 650, 651, 653
 Richards, V. 1120
 Richardson, A. P. 982
 Richter, H. 1009
 Rieders, F. 1322, 1323
 Riedler, G. 1121
 Rietbrock, N. 5, 1122, 1123, 1124, 1125
 Rinkel, M. 1126, 1127, 1128
 Ris, F. 1129
 Roach, M. K. 279, 281, 1130, 1131
 Robbins, B. H. 743, 1132
 Robert 1133
 Roberts, C. 1134
 Rodman, H. 1135
 Røe, O. 1136, 1137, 1138, 1139, 1140, 1141, 1142, 1143, 1144
 Rogers, W. A. 470
 Roggin, G. 655, 656, 658
 Rogina, V. 640
 Rohde, H. 1145
 Rojas-Ramirez, J. A. 572
 Rollins R. L., Jr. 1146
 Rombach, M. 1159
 Rondepierre, J. -J. 757
 Ropert, R. 757
 Rose, M. J. 268
 Röseler, P. 829, 1147
 Rosenbaum, M. 893, 1148
 Rosenfeld, G. 1149
 Rosenstein, R. 1333, 1334
 Rosenthal, S. M. 1150
 Ross, R. C. 165
 Roth, G. M. 1151, 1152
 Roth, J. A. 1153
 Röthlisberger, M. 1433
 Rouet, C. 1154
 Rouke, F. L. 927
 Rountree, C. B. 597, 602
 Roy, P. B. 1155
 Royer, R. 1156, 1157, 1158, 1159
 Rozé, C. 303
 Rozhnov, V. E. 1160
 Rożkowski, K. 721
 Rubin, A. L. 977
 Rubin, E. 786, 789, 894, 1161, 1162, 1163, 1164, 1165, 1166, 1167, 1168
 Ruckart, R. 294
 Rudolf, W. 1169
 Rummel, W. 1170
 Runeburg 1171
 Rungta, S. S. 291
 Runkevich, M. 1172
 Russek, H. I. 1173
 Rutenfranz, J. 1174
 Ryback, R. S. 150, 151, 1097, 1175, 1176, 1177
 Rydberg, S. 1178
 Rydberg, U. 497
 Sá Marques, M. M. 795, 796
 Saadeh, F. 287

- Saar, H. 1179
 Sachdev, K. S. 1180, 1181
 Säger, U. 1122
 Saidkasymov, T. 1182
 Salant, W. 1183
 Saldeen, T. 1184
 Salén, E. B. 1185
 Salmons, J. A. 943
 Sammalisto, L. 86, 1422
 Samochowiec, L. 1186
 Sand 1187
 Sandberg, F. 1188
 Sandler, R. 1189
 Sankar, B. 1190
 Sankar, D. V. S. 1190
 Santamaria, J. N. 1471
 Santesson, C. G. 1191
 Sassi, P. 274
 Sautet, J. 1192
 Sbertoli, C. 1193
 Scarlato, G. 1194
 Schambye, P. 1381
 Schelb, H. 1195
 Scherr, L. 977
 Schiefgen, W. 1196
 Schleyer, F. 375, 1197, 1198, 1199
 Schlick, B. von 746
 Schlierf, G. 1200
 Schlungs, H. 1201
 Schmelz, J. 371
 Schmid, P. 681, 682, 684
 Schmidt, G. 1202, 1278
 Schmidt, W. 1261, 1262
 Schmitz, B. 1405
 Schmitz, T. 1170
 Schneider, A. 1203
 Schoen, R. 1204
 Scholz, N. 837
 Scholz, R. 570
 Schreiner, G. E. 1205
 Schroeter, L. C. 1206
 Schulman, M. P. 444
 Schultz, J. D. 1207
 Schultz, R. E. 941
 Schüppel, R. 1208, 1209, 1210, 1211, 1212, 1213, 1214, 1215, 1216, 1217, 1218, 1280, 1281, 1284
 Schuth, W. 1219
 Schwartz, L. 781
 Schwarzmann, V. 1220
 Schwedtke, G. 1221
 Schwerd, W. 1448
 Scogin, J. T. 1222
 Scott, P. D. 1223
 Sebastianelli, A. 1224
 Seeberg, V. P. 1225
 Seevers, M. H. 1226
 Seidel, G. 1227, 1228, 1229, 1230, 1231
 Sellschopp, U. 1232
 Serantes, N. 744
 Serianni, E. 891, 1233, 1234
 Serrano, P. 1235
 Serusclat, F. 848
 Seydel, U. 136, 137
 Seydoux, J. 450, 451, 452, 453
 Sfikakis, P. 1236
 Shagass, C. 1237
 Sharma, J. D. 822
 Shea, J. 19, 393, 1207
 Sheard, C. 1151, 1152
 Shellenberger, T. E. 978
 Shelley, W. B. 1238
 Shepherd, M. 1239
 Sherlock, P. 1360
 Shimazu, R. 1240, 1241
 Shinaberger, J. H. 1242
 Shore, P. A. 176, 177
 Shorell, I. D. 1243
 Siegmund, B. 1244
 Siepmann, H. 1245, 1246
 Siew, S. 675
 Sigg, E. B. 1247
 Sigmund, W. 327
 Signorelli, S. 1248
 Silver, S. L. 176
 Simandl, J. 1249, 1250
 Simon, A. 187, 678, 1326, 1349
 Simon, P. 159
 Simon, W. 1251
 Simons, E. L. 643
 Simsch, A. 486, 1252
 Singh, J. M. 1253, 1254, 1255
 Sinitsyn, S. N. 1256, 1257
 Sirnes, T. B. 1258
 Sizemore, G. 649
 Sjövall, H. 1259
 Sloane, R. B. 206
 Slusher, N. 948
 Small, M. D. 1260
 Smart, R. G. 680, 1261, 1262
 Smetana, H. 1263
 Smilga, J. 1264
 Smillie, W. G. 1265
 Smith, A. A. 1266, 1267
 Smith, C. M. 1268
 Smith, H. 155
 Smith, H. W. 1269
 Smith, J. W. 1270

- Smith, M. E. 1271, 1272, 1273
 Smith, R. B., Jr. 537
 Smith, R. H. 392
 Smith, S. E. 1274
 Smolenski, U. 607
 Smorlesi, L. 1494
 Smyth, H. F. 1275
 Smyth, H. F., Jr. 1275
 Snell, A. M. 846
 Söderström, N. 1276
 Soehring, K. 438, 519, 522, 634, 931, 1208, 1210, 1227, 1228, 1229, 1230, 1277, 1278, 1279, 1280, 1281, 1282, 1283, 1284, 1285
 Sofronov, N. S. 1286
 Sögnen, E. 1287
 Solms, H. 1288
 Solms, W. 1289
 Solodkowska, W. 840
 Solomon, P. 864
 Somers, G. F. 1290
 Somogyi, E. 1065
 Souchart, M. 303
 Southard, F. D. 1012
 Spann, W. 329
 Spector, E. 1110
 Spranger, M. 1291
 Spreng, R. W. E. 1292
 Sroka, K. H. 1293
 Stacchini, C. 1294
 Staehelin, J. E. 1295
 Staffieri, J. J. 210
 Stančák, A. 1296
 State of California, County of Santa Clara, Department of the District Attorney, Laboratory of Criminalistics 1297
 State of California, Department of the Highway Patrol 1298
 Staub, H. 1299, 1300
 Stecher, W. 568
 Steiner, F. A. 1062
 Steinhoff, C. 1301
 Steinhoff, D. 1302
 Stenger, E. G. 1342, 1343
 Stepanov, A. V. 1303
 Stephan, L. 1304
 Stern, L. 1305
 Stern, M. M. 1306
 Stessel', T. A. 1307, 1308
 Stevens, H. 1309
 Stewart, J. 573
 Stewart, R. D. 1310
 Steyn, D. G. 1311
 Stiefbold, E. -G. 1312
 Štikar, J. 593
 Stoelting, V. 1036
 Stolman, A. 1313
 Stolman, S. 94
 Stone, G. C. 637
 Stone, M. M. 919
 Storelli, F. 302
 Störmer, A. 1314
 Straccia, F. A. 184
 Streichenberger, G. 1315
 Streller, I. 1227, 1228, 1230
 Streller, J. 1210
 Strongin, E. I. 1316, 1317
 Styszewska, H. 530
 Sugihara, N. 1318
 Sullivan, G. A. 1319
 Sulser, F. 1320, 1321
 Sunshine, I. 1322, 1323
 Suomalainen, H. 1422
 Süß, W. 1324
 Sutherland, R. B. 920
 Sutherland, V. C. 187, 679, 1325, 1326, 1349
 Sutter, W. 1068
 Svedin, C. -O. 1327
 Sykowski, P. 581
 Szücs, J. 1328
 Tacker, M. 1329
 Tainter, M. L. 980, 981
 Takabatake, E. 80, 81
 Takahasi, K. 79
 Takemori, A. E. 1330
 Takki, S. H. 152
 Talbert, W. 649
 Tamburrini, N. 1331
 Tammaro, A. E. 237, 238
 Tammisto, T. 1332
 Tang, P. C. 1333, 1334
 Tara, M. S. 1335
 Tarpey, R. D. 927
 Tarsitano, F. 1336
 Taylor, H. M. 950
 Taylor, J. D. 1337, 1476
 Teare, R. D. 1338
 Tedoldi, A. 911
 Teger, A. I. 660, 1339
 Teisinger, J. 1340, 1341
 Tenen, S. S. 703, 704
 Tephly, T. R. 1436, 1437
 Theilgaard, A. 1112, 1113
 Theobald, W. 1342, 1343
 Theorell, H. 147, 1344
 Thimann, J. 1345
 Thiry, U. 1346
 Thomas, B. H. 1465
 Thomas, J. R. 1347

- Thompson, M. J. 465
 Thompson, T. O. 1348
 Tipton, D. L., Jr. 187, 1349
 Tirri, R. 1350, 1420
 Többen, H. 1351
 Tobon, F. 655, 656, 657, 658
 Toll, N. 1352
 Tomaszewska, Z. 1353
 Tomits, G. 1354
 Tommasino, P. O. 210
 Tønning, D. J. 172, 1355
 Töpken, A. 1356
 Torka, J. 1357
 Torkelson, T. R. 1310
 Torres Orrego, R. 1358
 Tourtellotte, W. W. 1359
 Tracey, J. P. 1360
 Tracy, C. H. 255
 Trahan, P. 1254
 Traquair, H. M. 1361
 Travell, J. 489, 490
 Trenholm, H. L. 261, 264, 1362, 1363, 1463, 1464, 1465
 Truitt, E. B., Jr. 341, 1364, 1424
 Tryding, N. 379
 Tuğrul, S. 666
 Tunnicliffe, F. W. 183
 Tuohy, E. B. 502
 Tuovinen, P. I. 1365
 Turkel, H. W. 582
 Tygstrup, N. 805
 Tyler, V. E., Jr. 1468
 Uchermann, R. 1366
 Uhr, L. 874
 Ukai, M. 1367
 Umiker, W. 1368
 Unal, M. O. 1444
 Ungerleider, J. T. 1369
 Urechia, C. -I. 1370
 Uzawa, H. 1200
 Vachetta, A. 1371
 Vaillant, G. E. 1372
 Vaille, C. 303
 Valentino, C. 1373
 Valicenti, J. F., Jr. 1374
 Valiente, S. 209
 Van Dyke, R. 1499, 1500
 Vapaatalo, H. 992, 1375, 1376
 Varela Rodriguez, J. 380, 382
 Varley, M. 214
 Varney, D. H. M. 1377
 Vartiainen, O. 355, 1382
 Vassanelli, P. 663
 Vatteteau 1378
 Veale, W. L. 1379
 Veldstra, H. 1380
 Vendsborg, P. B. 1381
 Veneroni, E. 923
 Venho, E. V. 355, 1382
 Venho, I. 355, 1382
 Venturini, M. 1234
 Verron, G. 1383
 Videla, L. 1384
 Vifliantsev, N. M. 1303
 Villiaumey, M. J. 1385
 Vincenzi, L. 1386
 Vinnick, L. 1189
 Virolainen, E. S. 152
 Vlk, H. 1387
 Vogel, G. 1388, 1389
 Voigt, G. 379, 1259
 Voisin, J. 1096
 Voith, K. 1390
 Vollmer, H. 1391
 Volovik, V. M. 1392
 Von Hagen, D. S. 1393
 Votava, Z. 1394
 Vyas, D. S. 822
 Vyskočil, J. 107
 Wacker, W. E. C. 1053, 1395
 Wade, D. J. 1396
 Wagner, H. 614
 Wagner, H. -J. 701, 1397, 1398, 1399, 1400, 1401, 1402, 1403, 1404, 1405, 1406, 1407, 1408
 Wagner, K. 1407, 1408
 Wahlström, G. 1409, 1410, 1411, 1412
 Wahren, H. 1413
 Waisman, G. 744
 Walczak, J. 530
 Walker, G. 1414
 Walker, J. M. 143
 Walker, J. T. 411
 Wallace, G. B. 1415
 Wallace, J. 151
 Wallgren, H. 86, 1416, 1417, 1418, 1419, 1420, 1421, 1422, 1423
 Walsh, M. J. 299, 1424
 Walter, U. 1425, 1426
 Walther, R. 975
 Walzl, E. M. 845
 Wambsganss, E. 1427, 1428
 Wandrey, D. 1429
 Wangel, J. 1430
 Warburg, O. 1431
 Waris, E. 1432
 Warson, M. D. 397
 Wartburg, J. -P. von 1433
 Wasik, A. 1434, 1435

- Watkin, G. S. 644
Watkins, W. D. 1436, 1437
Watts, J. 1320, 1321, 1438
Wax, J. 1439
Wayne, E. J. 1440, 1441
Weatherall, M. 641
Weatherby, J. H. 1442
Webb, W. R. 304, 1443, 1444
Weber, J. 1445
Wei, E. 1446, 1447
Weinig, E. 1448
Weisburger, J. H. 1491
Weiss, B. 1449, 1450
Weiss, G. B. 1451
Weiss, L. R. 1452
Weiss, S. 821
Weissenberger, R. 1453
Welch, C. S. 257
Wells, H. S. 465, 740, 1454
Wendt, B. 1389
Werkgartner, A. 1455
Werner, H. W. 1456, 1457
Wexler, D. 864
Weymouth, R. J. 1487
Weyrich, G. 1458, 1490
Whisnant, C. L. 249
White, R. L. 1459, 1460
Whitney, D. D. 1461
Whittlesey, P. 1462
Wiberg, G. S. 261, 264, 1362, 1363, 1463, 1464, 1465
Widerlöv, E. 1412
Widmark, E. M. P. 1466, 1467
Wiener, K. 698, 700
Wier, J. K. 1468
Wikler, A. 443, 1469
Wilbur, D. L. 1470
Wilhelmi, G. 1343
Wilkinson, P. 1471
Willard, P. W. 1472
Williams, F. E. 547
Williams, H. S. 164
Williams, M. B. 1473
Willoquet, P. 224, 225, 227
Wilson, A. S. 1474, 1475
Wilson, L. 1337, 1476
Wilson, M. 314, 315
Wilson, R. H. 1477
Winfield, D. L. 1478
Wing, R. 741, 742
Winkler, K. 805
Winsor, A. L. 1317
Wiseman, B. D. 771
Wisner, P. -H. 1479
Wójcicki, J. 1186, 1480
Wolf, M. 1481
Wolff, H. G. 1482
Wölkart, N. 1483
Wolters, H. G. 1283
Wong, L. C. K. 1446, 1447
Wood, C. A. 1484
Woodhouse, S. W. 1485
Woods, D. P. 641
Wooles, W. R. 1486, 1487, 1488
Wright, F. 1489
Wuermeling, H. B. 1490
Wyatt, J. P. 920
Xavier, R. 276
Yamamoto, R. S. 1491
Yamanaka, Y. 299
Yen-Koo, H. C. 297
Yonetani, T. 1344
Young, J. V. 532
Yuguchi, T. 618
Zaccala, M. 923
Zaffiri, O. 1492
Zaikonnikova, I. V. 1092
Zakrividoroga, S. P. 1493
Zamcheck, N. 1260
Zampi, G. 1494
Zange, M. 1218
Zare, N. C. 967
Zatman, L. J. 755, 1495
Zbinden, G. 1496
Zeive, P. 655, 656, 658
Zimmermann, E. 1497
Zipf, H. F. 1498
Zirkle, G. A. 1499, 1500
Zohman, B. L. 1173
Zunin, L. 321
Zupko, A. G. 1206

DRUG INDEX

- Acacia – C
561
- A.C.E. – N
(alcohol + chloroform + diethylether)
1498
- Acccarbromal – P — Sedamyl
1319
- Acepromazine maleate – C — Soprontin
569
- Acetaldehyde – C
215, 281, 349, 543, 817, 839, 936, 937, 1271,
1498
- Acetanilide – C — Antifebrin
523, 540, 579, 634, 1089
- Acetone – R
925, 1104, 1307, 1308
- Acetophenetidine *See* Phenacetin
- Acetyl strophanthidin – N
431, 1036
- Acetylaminoantipyrine – N
1209
- Acetylcholine – R
823, 1128, 1180, 1181, 1191, 1419, 1451, 1480
- Acetyl- β -methylcholine *See* Methacholine
chloride
- Acetylsalicylic acid – C — Aspirin
45, 58, 62, 72, 85, 91, 158, 164, 267, 295, 324,
345, 368, 369, 425, 501, 540, 619, 748, 785,
924, 925, 1028, 1032, 1066, 1089, 1201, 1203,
1356, 1366, 1425, 1426, 1453, 1481, 1482,
1498
- Acevaltratum – N
364
- Actemin *See* Amphetamine
- ACTH – P
865
- Activit – N
(caffeine + glucose + fructose)
1304
- Adenosine phosphate – P — Adenylic acid
816
- Adenosine triphosphate – C
1206
- Adenylic acid *See* Adenosine phosphate
- Adrenaline – P — Epinephrine, Epinephrine
hydrochloride, Suprarenin
62, 70, 102, 216, 234, 322, 417, 664, 674, 839,
891, 983, 1100, 1126, 1127, 1128, 1180, 1267,
1364, 1420, 1453, 1480
- Aktedron *See* Amphetamine
- Alanine – C
816
- dl*-Alanine – C
500
- Alcohol minus – N — ALMI
(honey + fructose + glucose + carbonic
acid)
1095
- Aldehydes – N
2, 954, 1243
- Alkaline phosphate – N
1367
- Allecur *See* Clemizole
- Allobarbitol – R — Dial
217, 219
- Alloxan – C
542
- Allyl alcohol – C
698, 700
- Allyl formate – N
1220
- Allyl isosulfocyanate *See* Allyl isothiocyanate
- Allyl isothiocyanate – C — Allyl isosulfocyanate
1186
- 2-(4-Allyl-2-methoxy-phenoxy)-*N,N*-
-diethylacetamide – C — G 29505
1342
- N*-Allylnormorphine hydrobromide *See*
Nalorphine
- Allypropymal *See* Aprobarbitol
- ALMI *See* Alcohol minus
- Altafur *See* Furmethonol
- Amidopyrine *See* Aminophenazone
- Amidopyrine-4-aminoantipyrine – N
1209
- Aminazine *See* Chlorpromazine
- Aminoacetic acid – R — Glycine
542, 750
- 4-Aminoantipyrine *See* Ampyrone
- 1-(*m*-Aminobenzenesulfonyl)-3-butylurea – C —
SB-1
128
- p*-Aminobenzoic acid – C
818, 1479
- γ -Amino-butyric acid *See* 4-Aminobutyric acid
- 4-Aminobutyric acid – C — γ -Amino-butyric
acid
431, 527, 1149
- Aminoheptane – N
762, 765

2-Amino-5-nitrothiazole – C

695

Aminophenazone – R — Amidopyrine, Aminopyrine, Dimethylaminophenazone, Pyramidon

15, 158, 362, 368, 369, 520, 552, 553, 578,
654, 783, 784, 785, 925, 996, 1046, 1047,
1066, 1089, 1201, 1208, 1211, 1214, 1230,
1239, 1282, 1356, 1370, 1435, 1453, 1481,
1498

Aminophenazone + Phenylbutazone – C —

Irgapyrin

362, 619, 751, 753, 783, 784, 785, 866, 1066,
1080, 1118, 1397, 1398, 1498

Aminophenol – N

199, 1089, 1498

m-Aminophenol – C

737

o-Aminophenol – C

737

p-Aminophenol – C

623, 1221

Aminopyrine *See* Aminophenazone

Aminosalicylic acid – P

530

3-Amino-1,2,4-triazole – N

787, 788, 973, 974, 1131, 1272

Amitriptyline – R — Tryptizol

245, 547, 553, 573, 745, 792, 800, 810, 867,
878, 879, 881, 882, 885, 886, 887, 1039, 1258,
1498

Ammonia – C

240, 256, 471, 483, 484, 550, 917, 1120, 1378,
1485

Ammonia water – C

250, 352, 949

Ammonium acetate – C

95, 844

Ammonium bromide – C

523

Ammonium carbonate – C

169, 193, 1134

Amobarbital – P — Amobarbital sodium,

Amylobarbital, Amylobarbitone,
Amylobarbitone sodium, Amytal, Barbamil,
Dorlotin, Eunotal, Pentymal, Sodium amytal
23, 24, 46, 47, 155, 197, 245, 261, 264, 395,
540, 554, 717, 759, 814, 818, 877, 961, 970,
1073, 1093, 1154, 1184, 1188, 1237, 1274,
1286, 1354, 1392, 1448, 1463, 1498

Amobarbital sodium *See* Amobarbital

Amphenone *See* Amphenone B

Amphenone B – C — Amphenone

419

Amphetamine – R — Actemin, Aktedron,

Amphetamine sulfate, Benzedrine, Benzedrine
sulfate, Ortedrine, Phenamine,

1-Phenyl-2-aminopropane

68, 72, 76, 87, 130, 220, 247, 280, 281, 282,
308, 398, 429, 463, 495, 496, 498, 513, 539,
540, 592, 674, 760, 771, 815, 816, 839, 876,
983, 985, 1105, 1106, 1107, 1126, 1127, 1128,
1149, 1160, 1190, 1226, 1261, 1262, 1269,
1284, 1336, 1337, 1372, 1420, 1449, 1456,
1457, 1470, 1476

d-Amphetamine *See* Dexamphetamine sulfate

Amphetamine sulfate *See* Amphetamine

Ampyrone – R — 4-Aminoantipyrine

1211, 1212, 1213

Amyl alcohol – C

2, 98, 937, 1054, 1175, 1433

Amylobarbital *See* Amobarbital

Amylobarbitone *See* Amobarbital

Amylobarbitone sodium *See* Amobarbital

Amytal *See* Amobarbital

Analeptics – N

1219, 1229

Analgesics – N

121, 133, 167, 403, 500, 673, 680, 765, 807,
869, 1108, 1229, 1283, 1297, 1400, 1401, 1404,
1408, 1430, 1498

Anesthetics – N

167, 520, 956, 1154, 1161, 1498

Anethole – C

159

Angiotensinamide – R

160

Aniline – C

199, 354, 737, 769, 991, 1063, 1089, 1167,
1168, 1221, 1498

Aniline derivatives – C

1498

Animal charcoal *See* Carbon, amorphous

Antabuse *See* Disulfiram

Antibiotics – N

185, 1219, 1229, 1301, 1498

Anticholinergics – N

1297

Anticoagulants – N

39, 403, 431, 1121

Antidepressants – N

680, 683, 1111, 1406

Antidiabetics – N

403, 500, 807, 1283, 1297, 1298, 1498

Antiepileptics – N

167, 1229, 1283, 1429, 1498

Antifebrin *See* Acetanilide

- Antihistamine – N
1366
- Antihistamines – N
43, 44, 246, 332, 403, 431, 460, 680, 807, 861,
869, 1239, 1283, 1297, 1408, 1429, 1441, 1498
- Antihypertensives – N
133
- Antihypnotics – N
1498
- Antiinfectives – N
1297
- Antimony – C
142, 334
- Antipyretics – N
349, 375, 673, 680, 807, 869, 1034, 1301, 1430
- Antipyrine *See* Phenazone
- Antirheumatics – N
1498
- APC – P — Thomapyrine
(acetylsalicylic acid + caffeine + phenacetin)
552, 553, 584, 1066
- Apomorphine – C
247, 670, 760, 816, 994, 1288
- Apragon – N
376
- Aprobarbital – P — Allypropymal, Numal
243, 907, 908, 909
- Apronal – C — Sedormid
398, 783, 784, 785, 1046, 1047
- Arsenic – C
202, 401, 412, 1011, 1293, 1497, 1498
- Arsenic iodide *See* Arsenic triiodide
- Arsenic triiodide – C — Arsenic iodide
813
- Ascorbic acid – R — Cebione, Redoxon, Vitamin
C
139, 376, 542, 620, 688, 1469
- Aspirin *See* Acetylsalicylic acid
- Ataractics – N
133, 349, 403, 500, 1111, 1297, 1435
- Atebrin *See* Mepacrine
- Atosil *See* Isopromethazine
- Atropine – C — Methyl atropine
194, 216, 339, 353, 356, 621, 694, 816, 891,
901, 930, 956, 983, 1154, 1191, 1349, 1364,
1419, 1492, 1498
- Atropine methyl bromide – C
1077
- Atropine methyl nitrate – C — Eumydrine
102
- Atropine sulfate – C
1126, 1127, 1128
- Aureomycin *See* Chlortetracycline
- Azacyclonol – R
93, 506, 762
- Azides – N
405, 1131
- Bacchantyn – N
(combination drug)
1481
- Bampine – R — Soventol
1405
- Barbamil *See* Amobarbital
- Barbipyrine – C — Veramon
925
- Barbital – R — Barbital sodium, Barbitone,
Diemal, 5,5-Diethylbarbituric acid, Medinal,
Sodium barbital, Sodium veronal, Veronal,
Veronal sodium
25, 190, 192, 217, 219, 226, 261, 264, 278,
336, 368, 369, 561, 564, 593, 759, 825, 827,
861, 870, 907, 908, 909, 924, 976, 983, 1025,
1026, 1027, 1028, 1044, 1090, 1215, 1231,
1274, 1278, 1356, 1409, 1411, 1448, 1453,
1463, 1481, 1493
- Barbital sodium *See* Barbital
- Barbitone *See* Barbital
- Barbiturates – N
9, 32, 40, 41, 46, 47, 53, 57, 58, 66, 105, 121,
133, 167, 175, 191, 196, 199, 218, 221, 222,
223, 228, 229, 246, 249, 283, 314, 315, 330,
343, 346, 347, 348, 351, 358, 432, 443, 451,
520, 521, 527, 534, 570, 571, 593, 595, 617,
619, 628, 674, 680, 693, 717, 718, 725, 765,
784, 807, 839, 861, 887, 910, 916, 925, 929,
965, 970, 985, 1003, 1067, 1070, 1079, 1100,
1111, 1118, 1164, 1184, 1202, 1239, 1258,
1261, 1262, 1269, 1280, 1281, 1282, 1298,
1323, 1338, 1380, 1404, 1406, 1429, 1430,
1440, 1441, 1448, 1464, 1465, 1498
- Barbituric acid – C
546, 1277, 1401
- Barbituric acid derivatives – C
23, 24, 52, 270
- Bavarin 404 – N
174, 373, 374, 775, 1481
- Baycain *See* Tolycaine
- Bayer E 39 *See* Inproquone
- Baytinal *See* Buthalital sodium
- Beer – N
48, 51, 59, 83, 91, 123, 145, 207, 326, 327,
331, 333, 367, 370, 372, 432, 485, 521, 541,
551, 745, 747, 767, 784, 785, 798, 828, 855,
897, 911, 920, 929, 942, 946, 988, 1007, 1025,
1026, 1040, 1048, 1050, 1051, 1055, 1066,
1104, 1108, 1119, 1219, 1235, 1388, 1389,
1414, 1445, 1461, 1481, 1498

- Belladonna – N
153
- Bellafoline – N
1345
- Bellergal – N
(bellafoline + ergotamine tartrate + phenobarbital)
552, 553, 1066, 1498
- Bemegride – R — Eukraton
1357
- Benactyzine – R — Finalin
248, 588, 664, 762, 785
- Benthiazide – N
695
- Benzedrine *See* Amphetamine
- Benzedrine sulfate *See* Amphetamine
- Benzene – C
447, 1307
- Benzocaine – P
780
- Benzopyrene – C — Benzo- α -pyrene,
3,4-Benzpyrene
1110, 1130, 1163
- Benzo- α -pyrene *See* Benzopyrene
- Benzoquinamide *See* Benzquinamide
- 3,4-Benzpyrene *See* Benzopyrene
- Benzquinamide – R — Benzoquinamide
424, 426, 601, 676, 1062, 1492
- Bicarbonates – N
13, 769
- Billroth mixture – N
(chloroform + ether + alcohol)
1498
- Biphenyl – C
281
- Bismuth – C
1134, 1484
- Bismuth nitrate – C — Bismuth subnitrate
1305
- Bismuth subnitrate *See* Bismuth nitrate
- Bradykinin – C
1159
- Bromethol – P — Narcolan, Tribromethanol
9, 1286, 1498
- Bromides – N
595, 916, 1146
- Brompheniramine – R — Ilvin
1405, 1486
- Bromureide – N
23, 24
- Buclizine – P
495, 496
- Buclozine – N
492, 493
- Butabarbital *See* Secbutabarbital
- Butallylonal – P — Pernocton
1413
- Butamin – C — Tutocaine
101, 1010
- Butanilicaine – P — Hostacaine
552
- 1-Butanol – C — *n*-Butyl alcohol
138, 776, 1054, 1057, 1157, 1433
- 2-Butanol – C — *i*-Butyl alcohol
98, 138, 937, 1175
- Butazolidin *See* Phenylbutazone
- d*-Butazolidin *See* Phenylbutazone
- Butazolidine *See* Phenylbutazone
- Butethal – P — Butobarbital, Neonal, Soneryl
155, 411, 759, 1448, 1498
- Buthalital sodium – P — Baytinal
1076
- Butobarbital *See* Butethal
- Butriptyline – P
1390
- i*-Butyl alcohol *See* 2-Butanol
- n*-Butyl alcohol *See* 1-Butanol
- Butylchloral hydrate – P — Butylchlorhydrate
1057, 1498
- Butylchlorhydrate *See* Butylchloral hydrate
- Butylethylmalonylurea – N
1016, 1018, 1019, 1020, 1021
- Butyraldoxime – N
704
- n*-Butyraldoxime – N
199, 433, 683, 695, 703, 781, 1082, 1498
- Butyrolactone – C
948
- Butyrophenone – C
195
- Cafaspin – N
1108
- Caffeine – C
27, 28, 85, 140, 213, 216, 322, 370, 374, 375,
415, 422, 428, 429, 430, 432, 499, 504, 528,
539, 540, 545, 566, 598, 620, 629, 674, 723,
724, 730, 734, 746, 782, 791, 807, 812, 816,
865, 924, 925, 928, 942, 964, 966, 983, 984,
985, 989, 996, 1008, 1034, 1041, 1059, 1063,
1078, 1084, 1153, 1174, 1183, 1195, 1197,
1204, 1232, 1400, 1445, 1453, 1498
- Calcium – C
890
- Calcium bromide – C
764
- Calcium carbimide – P — Calcium cyanamide,
Dipsan

- 199, 349, 624, 625, 638, 683, 705, 726, 1033,
1034, 1067, 1346, 1364, 1498
- Calcium chloride *See* Calcium chloride,
anhydrous
- Calcium chloride, anhydrous – **C** — Calcium
chloride
22, 477, 1367
- Calcium cyanamide *See* Calcium carbimide
- Calcium ions – **N**
1449
- Calcium mesoxalate – **C**
962, 963
- Calcium nitrate – **C**
563
- Calomel *See* Mercurous chloride
- Camphor – **C**
193, 1204
- Cannabis – **C** — Marihuana
637, 675, 836, 837, 849, 850
- Captodiamine – **P** — Captodiamine, Covatix
506, 553
- Captodiamine *See* Captodiamine
- Carbachol – **P** — Carbaminoyl choline
816, 818, 820, 1128, 1180
- Carbamazepine – **P** — Tegretol
389, 709, 832
- Carbaminoyl choline *See* Carbachol
- Carbaryl – **P**
1452
- 1-Carbethoxymethyl-5,5-diallylbarbituric acid – **N**
1188
- Carbethoxymethyl-diethyl-barbituric acid – **N**
1448
- Carbocaine *See* Mepivacaine
- Carbocloral – **P** — Ethyl trichloramate
506
- Carbogen – **N**
556
- Carbolic acid *See* Phenol
- Carbon, amorphous – **C** — Animal charcoal
500, 1080, 1157
- Carbon dioxide – **C**
375, 540, 542, 543
- Carbon disulfide – **C**
354, 1477
- Carbon monoxide – **C**
119, 144, 174, 298, 788, 829, 912, 1050, 1131,
1147, 1185, 1335, 1408, 1498
- Carbon tetrachloride – **C**
1, 20, 21, 26, 78, 96, 120, 257, 272, 273, 349,
354, 392, 409, 437, 440, 465, 470, 507, 532,
551, 558, 712, 740, 741, 742, 743, 770, 821,
838, 840, 842, 846, 857, 890, 914, 915, 920,
943, 952, 953, 977, 1034, 1085, 1132, 1150,
1263, 1265, 1275, 1309, 1310, 1360, 1365,
1368, 1446, 1447, 1454, 1498
- Carbonic acid – **C**
331
- Carbutamide – **P** — Nadisan, Nadison
105, 132, 208, 286, 420, 647, 749, 784, 785,
824, 962, 963, 1065, 1158, 1498
- Cardiazole *See* Pentetrazole
- Carnitine – **C**
498, 513
- Casein – **C** — Caseosan
1391
- Caseosan *See* Casein
- Caustic soda *See* Sodium hydroxide
- Cebione *See* Ascorbic acid
- Centrophenoxine *See* Meclofenoxate
- Cetavlon *See* Cetrimonium bromide
- Cetrimonium bromide – **R** — Cetavlon,
Cetyltrimethyl ammonium bromide
1419
- Cetyl alcohol *See* 1-Hexadecanol
- Cetylpyridine chloride *See* Cetylpyridinium
chloride
- Cetylpyridinium chloride – **R** — Cetylpyridine
chloride
1419
- Cetyltrimethyl ammonium bromide *See*
Cetrimonium bromide
- Chlormethiazole – **P**
161, 1216, 1217
- Chloral – **C**
104, 325, 523, 1091, 1315
- Chloral hydrate – **C**
7, 8, 9, 49, 50, 69, 108, 133, 145, 203, 204,
278, 279, 444, 472, 473, 474, 475, 651, 652,
653, 717, 764, 765, 1016, 1020, 1021, 1033,
1063, 1308
- Chloralose – **C**
721
- Chlorcyclizine – **P**
87, 495, 496, 677, 692, 1475, 1486, 1487, 1488
- Chlordane – **C**
692
- Chlordiazepoxide – **R** — Clopoxide, Librium,
Methaminodiazepoxide
161, 170, 189, 283, 292, 297, 319, 337, 338,
339, 340, 344, 364, 365, 402, 426, 432, 441,
452, 453, 467, 468, 493, 494, 495, 496, 521,
531, 554, 572, 587, 600, 602, 605, 676, 681,
682, 684, 685, 686, 718, 757, 762, 822, 834,
873, 880, 945, 946, 947, 971, 1042, 1066,
1068, 1155, 1184, 1223, 1285, 1352, 1496,
1498
- Chlormethiazole *See* Clomethiazole

Chlormezanone

- Chlormezanone – **R**
425
- Chlorobenzene – **C**
897
- 7-Chloro-2, 3-dihydro-1-methyl-5-phenyl-1H-1, 4-benzodiazepine hydrochloride-**N**—Ro 5-4556
131
- Chloroethane – **C** — Ethyl chloride
956
- Chloroform – **C**
35, 221, 222, 243, 257, 736, 852, 892, 956, 983, 1011, 1063, 1091, 1129, 1150, 1308, 1351, 1371, 1446
- p*-Chlorophenylalanine – **N**
1379
- Chlorophyllin – **N**
509
- Chlorpromazine – **P**
527
- Chloroquine – **R**
683, 1498
- Chlorothiazide – **P**
695
- Chlorpheniramine – **P** — Chlortrimeton
1206
- Chlorpromazine – **P** — Aminazine,
Chlorpromazine hydrochloride, Fenactil,
Largactil, Megaphen, Phenactyl, Thorazine
19, 51, 56, 93, 105, 115, 126, 166, 168, 176, 177, 185, 187, 195, 235, 248, 275, 284, 292, 341, 350, 357, 359, 365, 393, 394, 421, 423, 426, 429, 430, 432, 448, 452, 453, 467, 468, 491, 492, 493, 495, 496, 531, 535, 547, 573, 582, 597, 600, 631, 664, 676, 678, 679, 691, 714, 715, 717, 718, 725, 757, 761, 762, 763, 792, 801, 804, 848, 858, 880, 882, 883, 885, 887, 888, 898, 899, 900, 903, 904, 905, 923, 933, 947, 989, 1034, 1067, 1076, 1087, 1160, 1169, 1199, 1207, 1228, 1273, 1280, 1281, 1301, 1320, 1326, 1349, 1369, 1375, 1376, 1405, 1441, 1492, 1498, 1499
- Chlorpromazine hydrochloride *See* Chlorpromazine
- Chlorpropamide – **P**
42, 105, 146, 208, 209, 210, 414, 695, 744, 1064, 1100, 1156, 1157, 1158, 1235, 1248, 1364, 1498
- Chlorprothixene – **R** — Truxal
152, 161, 188, 396, 547, 573, 1498
- Chlortetracycline – **P** — Aureomycin
366, 654, 1076
- Chlortrimeton *See* Chlorpheniramine
- Choko-milk – **N**
(cocoa + sugar + glucose + milk components)
1304
- Cholesterol – **C**
1206
- Choline – **R**
819
- Cibalgin – **N**
362, 584, 1028, 1046, 1047, 1448, 1498
- Cinchocaine chloride – **P** — Percaine
101, 1010
- Citrated calcium carbimide – **C**
810, 1082, 1100
- Clarmil *See* Tolboxane
- Clemizole – **P** — Allecur
604, 1405
- Cliradon *See* Ketobemidone
- Clomethiazole – **P** — Chlormethiazole
178, 1327, 1375, 1376, 1394
- Cloventhixol – **R**
1498
- Clopoxide *See* Chlordiazepoxide
- CNS drugs – **N**
1056
- Cobalt chloride – **C** — Cobaltous chloride
311
- Cobaltous chloride *See* Cobalt chloride
- Coca-cola – **N**
942, 1078
- Cocaine – **C**
58, 103, 313, 594, 850, 865, 1010, 1063, 1071, 1086, 1264, 1351
- Codeine – **C**
23, 24, 62, 421, 423, 426, 429, 430, 493, 600, 926, 1498
- Coffee – **N**
59, 95, 117, 368, 401, 528, 536, 713, 1269, 1316, 1317
- Commotional – **N**
(papaverin hydrochloride + caffeine + phenylethylbarbituric acid + phenacetin + dimethylamino-phenyldimethylpyrazolone)
1405
- Congeners – **N**
3, 88, 110, 150, 184, 212, 230, 244, 287, 288, 289, 290, 317, 318, 380, 381, 382, 383, 391, 400, 510, 511, 537, 542, 610, 659, 660, 687, 767, 935, 938, 940, 941, 967, 1097, 1177, 1339, 1461, 1474
- Contra – **N**
(coffee extract + dextrose + ammonium carbonate + vitamin B₁ + sodium carbonate + chlorophyllin + magnesium azulen)
374, 533, 734, 775, 1040, 1481

- Coprinus atramentarius – N
109, 121, 252, 253, 259, 262, 410, 431, 446,
469, 525, 606, 639, 683, 729, 783, 798, 1067,
1080, 1119, 1249, 1250, 1324, 1468
- Coprinus comatus – N
469
- Coramine *See* Nikethamide
- Corazol *See* Pentetrazole
- Cortisol *See* Hydrocortisone
- Cortisone – R
362, 701, 839, 1100, 1357
- Cortisone acetate – C — Cortone
865
- Cortone *See* Cortisone acetate
- Coumarin – C — Cumarin
1314
- Covatix *See* Captodiamine
- Cumarin *See* Coumarin
- Curare *See* Tubocurarine chloride
- Cyanamide – C
575
- Cyanamides – C — Zyanamid
397, 500, 544, 576, 626, 783, 1063, 1293, 1498
- Cyanocobalamin – R — Vitamin B₁₂
205
- Cycliton *See*
N,N-Diethyl-3,5-dimethyl-4-
isoxazolecarboxamide
- Cyclizine chloride – P — Marzine
338, 339, 340, 596
- Cyclobarbitol – P — Phanodorm
571, 759, 1188, 1448
- Cyclohexane – C
1307
- Cyclopropane – R
9, 1154
- Cycloserine – R — *d*-Cycloserine
486, 530, 1252, 1498
- d*-Cycloserine *See* Cycloserine
- Cycrimine hydrochloride – R — Pagitane
hydrochloride
277
- Cysteine – C
542, 726, 1157
- Cystine – C
542
- Cytisine – N
730
- D-860 *See* Tolbutamide
- Deanol – P — Dimethylaminoethanol
54, 498, 513, 514
- Decamethonium iodide – P
1419
- Dehydrobenzperidol – P
527
- Demerol *See* Pethidine
- Depressants – N
53, 349, 431, 1441
- Desipramine – R — Desmethylinipramine,
Pertofran
547, 882, 887, 1321, 1343, 1438
- Desmethylocarnitine – N
54, 514
- Desmethylinipramine *See* Desipramine
- Desmethylopromazine – N
547
- 11-Desoxo-cortisone-acetate – N
139
- Desoxyephedrine *See* Methamphetamine
- 2-Desoxy-*d*-glucose – N
1065
- Dexamphetamine – P — Dexedrine,
Dextroamphetamine
54, 815, 984
- Dexamphetamine sulfate – C — Maxiton,
d-Amphetamine
180, 258, 294, 514, 515, 603, 612, 650, 809,
1190, 1267
- Dexedrine *See* Dexamphetamine
- Dexphenmetrazine *See* Phenmetrazine
- Dextran – P
1242
- Dextroamphetamine *See* Dexamphetamine
- Dextroenergen *See* Glucose
- Dextromoramide – P
333
- Dextropropoxyphene – P — *d*-Propoxyphene
105, 421, 423, 426, 429, 430, 600, 1498
- Dextrose *See* Glucose
- DFP *See* Dyflos
- Dial *See* Allobarbitol
- Diamorphine – P — Heroin
313
- Diazepam – R — Valium
235, 285, 307, 337, 338, 339, 340, 364, 365,
432, 572, 605, 621, 642, 754, 810, 834, 882,
887, 923, 946, 1007, 1067, 1258, 1366, 1375,
1376, 1394, 1492, 1498
- Diazepin derivatives – N
1408
- Dibenzepin – P — Noveril
531
- Dibenzothiazyl disulfide – N
16
- Didrovaltratum – N
364
- Diemal *See* Barbitol

Diethylamine barbiturates

Diethylamine barbiturates – C — Somnifene
1358

5,5-Diethylbarbituric acid *See* Barbitol

N,N-Diethyl-3,5-dimethyl-4-isoxazolecarboxamide
– C — Cycliton
574

Diethyldiphenylthiuram disulfide – N
16

Diethylene glycol – C
1054

Digitalis – C
353, 435, 523, 791, 802, 803, 1030

Dihydrobenzthiazide *See* Hydrobentizide

Dihydrochlorothiazide *See* Hydrochlorothiazide

Dihydrocodeinone *See* Hydrocodone

Dihydroergotamine – C
553, 901

Dihydromorphinone *See* Hydromorphone
hydrochloride

Dilantin *See* Phenytoin

Dilatol-quinine – N
376

Dilcoran 80 – C
1066

Dimenhydrinate – P — Dramamine
1333, 1334

Dimethocaine – P — Larocaine
101, 1010

Dimethyl sulfoxide – P — DMSO
133, 386, 387, 431, 520, 565, 709, 789, 830,
831, 833, 834, 854

Dimethylaminoethanol *See* Deanol

Dimethylaminophenazone *See* Aminophenazone
N-(γ -Dimethylamino-propyl)-iminodibenzylum
hydrochloride *See* Imipramine

Diminal – N
161

Dimorpholynylthiuram disulfide – N
16

m-Dinitrobenzene – C
459, 623, 1115

o-Dinitrobenzene – C
623

p-Dinitrobenzene – C
623

Dinitrobenzol – N
254, 1063

2,4-Dinitro-1-chlorobenzene – C
254, 1063

Dinitrocresol *See* Dinitro-*o*-cresol

Dinitro-*o*-cresol – C — Dinitrocresol,
4,6-Dinitro-*o*-cresol
232, 305, 375, 557, 626, 960, 1467

4,6-Dinitro-*o*-cresol *See* Dinitro-*o*-cresol

Dinitrocyclopentylphenol – N
233, 234

Dinitrophenol – N
121, 216, 232, 375, 399, 457, 500, 557, 626,
734, 769, 960, 979, 980, 981, 983, 991, 1063,
1453

2,4-Dinitrophenol – C
390, 540, 542, 622, 998, 1381, 1384, 1467

2,5-Dinitrophenol – C
1467

2,6-Dinitrophenol – C
1467

Dinitrotoluol – N
991

Diphenhydramine – R
23, 24, 70, 604, 664, 1486

Diphenylhydantoin – C
61, 656, 1498

5,5-Diphenylhydantoin *See* Phenytoin

Diphosphopyridine nucleotide (DPN) *See*
Nicotinamide-adenine dinucleotide

N,N'-Diphenyl-*p*-phenylenediamine – C
266

Dipsan *See* Calcium carbimide

Dipyridamole – R — Persantin
1405

Distilled spirits – N
3, 6, 43, 44, 50, 55, 57, 58, 62, 73, 79, 139,
150, 170, 174, 178, 184, 204, 206, 213, 230,
244, 260, 288, 289, 290, 308, 317, 318, 331,
358, 367, 372, 380, 381, 382, 383, 400, 411,
427, 428, 435, 437, 442, 471, 495, 496, 509,
511, 512, 525, 537, 541, 542, 551, 556, 585,
587, 589, 590, 596, 636, 659, 660, 670, 687,
711, 713, 726, 738, 743, 745, 754, 767, 773,
778, 781, 793, 798, 799, 822, 828, 842, 843,
849, 850, 853, 855, 873, 895, 907, 908, 909,
911, 917, 918, 920, 927, 934, 935, 936, 937,
938, 939, 940, 941, 943, 946, 949, 966, 967,
970, 984, 995, 1007, 1008, 1013, 1026, 1040,
1042, 1055, 1066, 1069, 1097, 1104, 1108,
1121, 1135, 1173, 1175, 1176, 1177, 1198,
1201, 1203, 1244, 1296, 1319, 1326, 1334,
1337, 1347, 1365, 1377, 1387, 1388, 1389,
1434, 1458, 1461, 1474, 1476, 1481, 1484,
1485, 1490, 1498

Disulfiram – P — Antabuse, Tetraethylthiuram
disulfide
16, 30, 105, 121, 133, 171, 199, 201, 207, 209,
234, 247, 248, 259, 262, 281, 284, 319, 341,
349, 397, 431, 500, 544, 626, 689, 695, 720,
729, 751, 753, 760, 761, 762, 763, 764, 765,
810, 866, 966, 1033, 1034, 1052, 1067, 1082,
1100, 1109, 1118, 1154, 1156, 1157, 1159,

Fentanyl

- 1193, 1249, 1250, 1288, 1299, 1300, 1364,
1366, 1385, 1397, 1398, 1406, 1408, 1468,
1498
- Diuretics – **N**
1297
- Dixyrazine – **C**
871, 872, 1075
- DMSO *See* Dimethyl sulfoxide
- Dolantin *See* Pethidine
- Dolviran – **N**
(acetylsalicylic acid + phenacetin + caffeine
+ phenobarbital)
1052, 1108
- Dominal forte *See* Prothipendyl
- Dopamine – **C**
299, 913, 1149
- Dorlotin *See* Amobarbital
- Dormison *See* Methylpentynol
- Doxapram – **R**
294
- Dramamine *See* Dimenhydrinate
- Droperidol – **R**
1492
- Dyflos – **P** — DFP
818
- Edetic acid – **R** — Tetracemin
1287
- Egressin *See* Thymyl isoamylcarbamate
- Elixir of barbital N.F. VII – **N**
1225
- Emetine – **C**
1288
- Emylcamate – **P** — 3-Methyl-3-pentyl carbamate
248, 761, 762
- Enibomal – **P** — Eunarcon
693, 1116, 1117
- Enzymes – **N**
1165, 1272
- Eocene – **N**
1328
- Ephedrine – **C**
53, 70, 237, 322, 431, 539, 674, 694, 791, 816,
1413
- Epinephrine *See* Adrenaline
- Epinephrine hydrochloride *See* Adrenaline
- Ergotamine – **R**
1066
- Ergotamine tartrate – **P** — Gynergen
1345
- Ervinin – **N**
1182
- Eserine *See* Physostigmine
- Esonium – **C**
1494
- Esters – **N**
291, 954, 1243
- Ethacrynic acid – **P**
323
- Ethamivan – **P**
294
- Ethchlorovynol – **P**
9, 75, 283, 731
- Ether – **C**
2, 4, 9, 14, 15, 35, 162, 179, 221, 222, 354,
453, 595, 766, 956, 983, 997, 1087, 1091,
1133, 1179, 1187, 1307, 1378, 1413, 1492
- Ethinamate – **R**
506
- Ethionamide – **P**
530
- Ethionine – **C**
81
- Ethyl acetate – **C**
512, 936, 937, 939, 1175
- Ethyl chloride *See* Chloroethane
- Ethyl formate *See* Triethyl orthoformate
- Ethyl trichloramate *See* Carbocloral
- Ethyl urethane *See* Urethane
- Ethylbutyl-barbituric acid – **N**
1014, 1015, 1448
- Ethylene glycol – **C**
149, 163, 303, 1053, 1069, 1395, 1450
- Ethylisoamylmalonylurea – **N**
1017
- Ethylmethylpentanoic acid carbamide – **N**
763
- 5-Ethyl-5-(1-methylpropyl)-2-thiobarbituric acid –
C — Inactin
929, 1076, 1498
- Eucaine B – **C**
101, 1010, 1351
- Eukraton *See* Bemegride
- Eumed – **N**
(caffeine + phenacetin + aspirin +
pyramidon)
584
- Eumydrine *See* Atropine methyl nitrate
- Eunarcon *See* Enibomal
- Eunoctal *See* Amobarbital
- Evipal *See* Hexobarbital
- Evipan *See* Hexobarbital
- Fargan *See* Promethazine
- Fenactil *See* Chlorpromazine
- Fencamfamin – **R** — Reactivan
1230
- Fentanyl – **P**
527, 1492

Ferricyanide

- Ferricyanide – **N**
1271
- Ferronat *See* Ferrous gluconate
- Ferrous gluconate – **P** — Ferronat
583
- Finalin *See* Benactyzine
- Fluoroacetates – **N**
614, 1359
- Fluothane *See* Halothane
- Fluphenazine – **P** — Fluphenazine
dihydrochloride, Permitil
327, 432, 773, 1498
- Fluphenazine dihydrochloride *See* Fluphenazine
- Flurothyl – **R**
159
- Folic acid – **C**
1123
- Formaldehyde – **C**
671, 1431
- Fructose – **C** — Laevoral, Laevosan, Levulose
375, 500, 618, 701, 723, 734, 752, 775, 807,
865, 960, 1100, 1206, 1219, 1288, 1384, 1481
- Fruit – **N**
536
- Fruit juice – **N**
136, 137
- Fumarates – **N**
769
- 2-Furaldehyde – **C** — Furfural
2, 98, 183, 537, 954, 1243
- Furaltadone *See* Furmethonol
- Furazolidone – **R** — Furoxon
207, 683, 695, 711, 1067
- Furfural *See* 2-Furaldehyde
- Furmethonol – **R** — Altafur, Furaltadone
255, 466, 911
- Furosemide – **P**
160
- Furoxon *See* Furazolidone
- Fusel oil – **C**
331, 478, 954, 1433
- G 29505 *See*
2-(4-Allyl-2-methoxy-phenoxy)-*N,N*-
-diethylacetamide
- Gardan – **N**
(aminophenazone +
acid sodium)
784, 1425, 1426, 1453, 1481, 1498
- Gardenal *See* Phenobarbital
- Gasoline – **C**
373
- Gelonida – **N**
(codeine + phenacetin + aspirin)
376, 552, 553, 1066
- Ginseng – **C** — Panax ginseng
889, 1318
- Gitoxigenin – **C**
975
- Gitoxin – **C**
975
- Glimid *See* Glutethimide
- Glucose – **C** — Dextroenergen, Dextrose
30, 165, 185, 7, 342, 369, 477, 500, 582, 769,
775, 807, 816, 892, 906, 930, 960, 966, 1014,
1015, 1088, 1100, 1206, 1232, 1242, 1287,
1288, 1325, 1358, 1459, 1460
- Glutamate sodium – **N**
715
- Glutamates – **N**
1325
- Glutamic acid – **R**
431, 542, 816, 1400
- Glutathione – **C**
542
- Glutethimide – **R** — Glimid
75, 488, 506, 862, 863, 1009, 1205, 1322, 1353
- Glybuthiazole – **R**
1156, 1158
- Glycerin *See* Glycerol
- Glycerol – **R** — Glycerin
750, 769, 805, 806, 835, 1484
- Glyceryl trinitrate – **C** — Nitroglycerin, Trinitrin
353, 354, 523, 791, 1293
- Glycine *See* Aminoacetic acid
- Gothania-antialcohol tablets – **N**
(combination drug)
371, 373, 374, 1445, 1481
- Guaiacol glyceryl ether *See* Guaiphenesin
- Guaiphenesin – **P** — Guaiacol glyceryl ether
1498
- Gynergen *See* Ergotamine tartrate
- Haloperidol – **R**
56, 235, 923, 1427, 1428
- Halothane – **P** — Fluothane
9, 328, 527, 992
- Heroin *See* Diamorphine
- 1-Hexadecanol – **C** — Cetyl alcohol
611
- Hexamethonium – **R**
1349
- Hexenal *See* Hexobarbital
- Hexobarbital – **R** — Evipal, Evipan, Hexenal,
Hexobarbitone
14, 15, 94, 135, 278, 356, 406, 407, 408, 438,
453, 535, 561, 571, 591, 1088, 1092, 1116,
1117, 1164, 1167, 1168, 1188, 1280, 1281,
1282, 1410, 1411, 1412, 1448, 1455, 1493,
1498

Isoprenaline

- Hexobarbitone *See* Hexobarbital
- Histamine – **C**
648, 721, 1180
- Hormones – **N**
30, 216
- Hostacaine *See* Butanilcaine
- Hydrobentizide – **P** — Dihydrobenzthiazide
695
- Hydrochloric acid – **C**
295, 613
- Hydrochlorothiazide – **R** —
Dihydrochlorothiazide
160, 695
- Hydrocodone – **R** — Dihydrocodeinone
617
- Hydrocortisone – **P** — Cortisol
185, 1444
- Hydrocyanic acid *See* Hydrogen cyanide
- Hydrogen cyanide – **C** — Hydrocyanic acid
1134, 1431
- Hydromorphone hydrochloride – **R** —
Dihydromorphinone
1498
- Hydroquinol *See* Hydroquinone
- Hydroquinone – **C** — Hydroquinol
737
- 4-Hydroxy phenazone – **N**
1211, 1212, 1213
- Hydroxyamphetamine – **P** — Paredrine
983, 1126, 1127
- Hydroxybutyrate – **N**
948
- Hydroxydione sodium – **P**
1216, 1217
- p*-Hydroxyephedrine – **C** — Suprifen
538
- N*-Hydroxy-*N*-2-fluorenylacetamide – **N**
1491
- 8-Hydroxyquinoline *See* 8-Quinolinol
- 5-Hydroxytryptamine *See* Serotonin
- Hydroxyzine – **R**
248, 292, 357, 421, 423, 426, 432, 492, 493,
495, 496, 503, 506, 597, 600, 676, 732, 733,
871, 872, 1498
- Hyoscine – **C**
153, 301, 413, 956
- Hypnophen – **N**
37, 908, 909
- Hypnotics – **N**
167, 375, 403, 460, 673, 680, 683, 807, 869,
1229, 1283, 1284, 1297, 1400, 1401, 1402,
1403, 1404, 1406, 1408, 1429, 1498
- Ilvin *See* Brompheniramine
- Imipramine – **R** —
N-(γ -Dimethylamino-
propyl)-iminodibenzylum
hydrochloride, Tofranil
235, 531, 547, 573, 757, 762, 792, 882, 1247,
1320, 1321, 1342, 1343, 1390, 1401, 1496,
1498
- Immenoctal *See* Secobarbital
- Inactin *See*
5-Ethyl-5-(1-methylpropyl)-2-thiobarbituric
acid
- Industrial poisons – **N**
500
- INH derivatives – **N**
1401, 1402, 1408
- Inproquone – **P** — Bayer E 39
1230
- Insidon *See* Opipramol
- Insulin – **C**
30, 105, 133, 158, 216, 234, 243, 257, 342,
375, 417, 499, 500, 535, 557, 734, 746, 775,
807, 892, 924, 925, 960, 962, 966, 979, 96,
1065, 1100, 1104, 1156, 1157, 1358, 1364,
1365, 1445, 1453
- Iodine natrium *See* Sodium iodide
- Iodoacetic acid – **C**
241, 769
- Iodobismital – **N**
982
- 4-Iodopyrazole – **N**
1178
- Ipecac – **C**
95
- Iproniazid – **P**
644, 1273, 1400
- Irgapyrin *See* Aminophenazone +
Phenylbutazone
- Iron – **C**
849, 1134
- Isoamyl alcohol *See* Isopentyl alcohol
- Isobutyl alcohol *See* 2-Methyl-1-propanol
- Isocarboxazid – **R**
245
- Isoniazid – **P** — Isonicotinic acid hydrazide,
Neoteben, Rimifon
133, 157, 185, 312, 333, 362, 485, 487, 500,
530, 568, 683, 693, 774, 783, 784, 785, 902,
1031, 1052, 1066, 1081, 1219, 1236, 1239,
1397, 1398, 1434, 1498
- Isonicotinic acid hydrazide *See* Isoniazid
- Isopentyl alcohol – **C** — Isoamyl alcohol
512, 936
- Isoprenaline – **P** — Isoproterenol
1267

Isopromethazine

- Isopromethazine – C — Atosil
1076
- Isopropamide *See* Isopropamide iodide
- Isopropamide iodide – R — Isopropamide
1427
- Isopropyl alcohol – C
864, 1054, 1439
- Isopropylallylmalonylurea – N
1017
- Isoproterenol *See* Isoprenaline
- Isovalerylaminophenazone – C — Neopyrine
1002
- Jatroneural *See* Trifluoperazine
- JB-516 *See* Pheniprazine
- Kanamycin – R
690
- Kemadrin *See* Procyclidine
- Kemithal *See* Thialbarbital
- Kerosene – C
611
- Ketamine – C
1216, 1217, 1218
- Ketobemidone – P — Cliradon
333
- Kö 592 – N
384
- L-67 *See* Prilocaine
- Lactic acid – C
769
- Lactose – C
843, 1337, 1476
- Laevoral *See* Fructose
- Laevosan *See* Fructose
- Largactil *See* Chlorpromazine
- Larocaine *See* Dimethocaine
- Lead – C
142, 354, 446, 851, 1063, 1171, 1293, 1498
- Lecithin – C
997
- Levomepromazine – P
757
- Levorphanol – P
1266
- Levulose *See* Fructose
- Librium *See* Chlordiazepoxide
- Lidocaine – P
1479
- Lime blossom extract – N
1378
- Liothyronine – P — 3,5,3-Triiodothyronine
121, 775, 1000, 1100
- Lithium compounds – N
972
- Lobeline – P
376, 791, 1076
- Local anesthetics – N
1492
- Luminal *See* Phenobarbital
- Lysergamide – N
762
- Lysergic acid diethylamide *See* Lysergide
- Lysergide – R — Lysergic acid diethylamide
54, 93, 177, 321, 498, 513, 514, 586, 841, 1268
- Magnesium hydroxide – C — Polysan
186
- Magnesium sulfate – C
247, 417, 477, 760, 1060, 1061, 1466
- Malate – N
769
- Malonate – N
769, 1418
- Mannitol – C
1492
- Marcoumar *See* Phenprocoumon
- Marihuana *See* Cannabis
- Marzine *See* Cyclizine chloride
- Maxiton *See* Dexamphetamine sulfate
- Mebhydrolin – R — Omeril
1405
- Meclozine *See* Meclozine
- Meclofenoxate – P — Centrophenoxine
765
- Meclozine – P — Meclozine
87, 495, 496, 1498
- Medinal *See* Barbital
- Megaphen *See* Chlorpromazine
- Melabon – N
1066
- Menthol – C
1251
- Mepacrine – R — Atebrin
362
- Mepazine *See* Pecazine
- Meperidine – P
527
- Mephedine *See* Pethidine
- Mephenesin – R — Tolserol
1292, 1319
- Mephenesin carbamate – C
82
- Mephenoxalone – C — Trepidone
432, 927
- Mepivacaine – R — Carbocaine
1479
- Meproamate – R — Miltown, Procalmadiol, Restenil
38, 93, 105, 188, 195, 214, 248, 249, 285, 292,

- 344, 350, 359, 363, 385, 421, 423, 426, 427, 429, 432, 441, 449, 492, 493, 494, 495, 496, 506, 552, 553, 561, 569, 582, 591, 592, 593, 597, 663, 664, 676, 681, 682, 684, 685, 686, 715, 725, 762, 763, 801, 804, 843, 874, 875, 894, 926, 929, 931, 932, 944, 945, 947, 971, 1002, 1034, 1042, 1068, 1087, 1111, 1112, 1113, 1114, 1166, 1169, 1194, 1258, 1285, 1322, 1357, 1430, 1441, 1498, 1500
- Mepyramine – **R** — Pyrilamine
1306
- Mercaptobenzothiazol *See*
2-Mercaptobenzothiazole
- 2-Mercaptobenzothiazole – **C** —
Mercaptobenzothiazol
16
- Mercuderamide – **R** — Neptal
1471
- Mercurous chloride – **C** — Calomel
153, 193, 523
- Mercury – **C**
142, 354, 446, 988, 1063, 1293, 1498
- Mercury iodide – **C**
813
- Mersalyl – **R** — Salyrgan
695
- Mescaline – **C**
1268
- Metahexamide – **R**
1158
- Methacholine chloride – **R** —
Acetyl- β -methylcholine
1077, 1128
- Methadone – **R** — Polamidone
127, 429, 430, 785, 1050, 1258, 1399, 1407, 1498
- Methaminodiazepoxide *See* Chlordiazepoxide
- Methamphetamine – **P** — Desoxyephedrine, Pervitin
182, 335, 372, 374, 375, 454, 504, 535, 562, 574, 609, 629, 674, 724, 734, 865, 925, 1066, 1078, 1083, 1174, 1244, 1295, 1336, 1453
- Methanol – **C** — Methyl alcohol
10, 11, 12, 13, 23, 24, 62, 76, 89, 90, 111, 116, 125, 138, 156, 172, 251, 269, 309, 358, 379, 418, 431, 479, 480, 481, 482, 649, 671, 672, 710, 755, 811, 864, 896, 936, 937, 1035, 1057, 1096, 1122, 1123, 1124, 1125, 1136, 1137, 1138, 1139, 1140, 1141, 1142, 1143, 1144, 1175, 1198, 1242, 1276, 1303, 1307, 1308, 1355, 1437, 1484, 1495
- Methaqualone – **R** — Revonal
161, 389, 709, 832, 1050
- Methdilazine – **R**
797
- Methionine – **R**
542
- Methitural – **R**
438, 591, 1498
- Methohexital – **R**
329, 621, 766
- Methophenobarbitone *See* Methylphenobarbital
- Methoxamine – **P**
648
- Methoxyflurane – **R**
9, 766
- Methyl alcohol *See* Methanol
- Methyl atropine *See* Atropine
- Methyl carbamate – **C** — Methyl urethane
524
- Methyl salicylate – **C**
864
- Methyl urethane *See* Methyl carbamate
- Methyl violet – **P**
455
- Methylaminoantipyrine *See* Noramidopyrine
- 3-Methylcholanthrene – **C**
692, 787, 1110
- Methyldopa – **R** — Presinol
1405
- Methylene blue – **P**
542, 1271, 1328
- Methylmercuric acetate – **N**
263
- 3-Methyl-3-pentyl carbamate *See* Emylcamate
- Methylpentynol – **R** — Domison, Oblivon
55, 72, 1009, 1206
- Methylpentynol carbamate – **C**
248, 761, 763, 1087
- Methylphenidate – **R** — Ritalin
592, 882, 887, 1000, 1222
- Methylphenobarbital – **R** —
Methophenobarbitone
1054
- 10-[3-(4-Methyl-1-piperaziny)-propyl]-
phenothiazine *See* Perazine
- N*-Methylpiperidyl-3-methylphenothiazine *See*
Pecazine
- 2-Methyl-1-propanol – **C** — Isobutyl alcohol
936
- 4-Methylpyrazole – **N**
147, 148
- l*-Methylxanthine – **N**
28
- Methypylone – **R** — Noludar
83, 127, 344, 506, 585, 684, 685, 686, 747,

Methyprylone

- 1048, 1049, 1050, 1051, 1052, 1068, 1478, 1496
 Metrazole *See* Pentetrazole
 Metronidazole – **R**
 121, 683, 1100
 Metrotonin – **N**
 (isoamylethylbarbituric acid +)
 1066
 Milk – **C**
 103
 Miltown *See* Meproamate
 Mirapront *See* Phentermine
 Mogadan *See* Nitrazepam
 Mogadon *See* Nitrazepam
 Monoamine oxidase inhibitors – **N**
 33, 115, 133, 134, 296, 349, 429, 430, 432, 1034, 1099, 1100, 1424, 1435
 Monochloroethanol – **N**
 1054
 Monofluoroethanol – **N**
 1054
 Morphine – **C**
 37, 58, 63, 100, 105, 123, 139, 145, 175, 216, 301, 355, 356, 358, 413, 421, 423, 426, 429, 430, 451, 452, 453, 521, 540, 620, 636, 668, 669, 674, 785, 839, 850, 910, 930, 956, 958, 959, 997, 1029, 1034, 1092, 1103, 1233, 1276, 1382, 1391, 1407, 1498
 Morphine hydrochloride – **C**
 99, 907, 908, 909, 951
 Morphine sulfate – **C**
 600
 Muscarine – **C**
 1191
 Mylepsin *See* Primidone
 Nadisan *See* Carbutamide
 Nadison *See* Carbutamide
 Nalorphine – **R** — *N*-Allylnormorphine
 hydrobromide
 450, 958, 959, 1330
 α -Naphthol *See* 1-Naphthol
 β -Naphthol *See* 2-Naphthol
 1-Naphthol – **C** — α -Naphthol
 737
 2-Naphthol – **C** — β -Naphthol
 737
 Narcolan *See* Bromethol
 Narcotics – **N**
 351, 436, 1197, 1429
 Natrium salicylicum – **N**
 1241
 Nembutal *See* Pentobarbital
 Neonol *See* Butethal
 Neopyrine *See* Isovalerylamino-phenazone
 Neostigmine – **P** — Prostigmine
 816, 818, 1128
 Neoteben *See* Isoniazid
 Neptal *See* Mercuderamide
 Neuroleptics – **N**
 167, 306, 683, 765, 794, 1229, 1288, 1400, 1406, 1435, 1498
 Neutragol – **N**
 (sugar + menthol + alcohol + plant extracts + nitrophenol)
 1425, 1426, 1481
 Nialamide – **R** — Niamid
 206, 235, 238, 432, 757, 762, 765, 913, 1498
 Niamid *See* Nialamide
 Nicoteben – **N**
 (neoteben + isonicotinaldehyde thiosemicarbazone)
 1066, 1230
 Nicotinamide – **R**
 618, 760, 816, 1358
 Nicotinamide-adenine dinucleotide – **C** —
 Diphosphopyridine nucleotide (DPN)
 1100, 1271, 1362
 Nicotine – **C** — Tobacco
 18, 59, 67, 97, 142, 143, 205, 293, 334, 361, 368, 375, 398, 529, 553, 667, 668, 669, 753, 783, 812, 859, 860, 866, 924, 985, 1001, 1004, 1063, 1098, 1151, 1152, 1173, 1232, 1251, 1361, 1372, 1400
 Nikethamide – **R** — Coramine
 154, 368, 376, 434, 559, 574, 620, 728, 791, 839, 861, 868, 986, 1038, 1094, 1245, 1246, 1377, 1387, 1413, 1453, 1456, 1457, 1489
 α -Nitranisol – **N**
 991
 Nitrazepam – **P** — Mogadan, Mogadon
 388, 389, 709, 832, 1366
 Nitrobenzene – **C** — Nitrobenzol
 199, 254, 354, 897, 1063
 Nitrobenzoic acid – **C**
 1498
 Nitrobenzol *See* Nitrobenzene
 Nitroglycerin *See* Glyceryl trinitrate
 Nitrophenol – **C**
 991, 1063, 1498
 Nitrotoluol – **N**
 991, 1063
 Nitrous oxide – **C**
 9, 328, 527, 538, 956
 NNR hormone – **N**
 (glucocorticoid of the adrenal cortex)
 865
 Noctal *See* Propallylonal
 Noludar *See* Methyprylone

Pentolinium tartrate

- Noradrenaline – **P** — Norepinephrine
238, 431, 648, 1424
- Noramidopyrine – **C** — Methylaminoantipyrine
1116
- Norepinephrine *See* Noradrenaline
- Norphenazone – **N**
1213
- Nortriptyline – **R**
422, 599, 867, 882
- Noveril *See* Dibenzepin
- Novocaine *See* Procaine
- Numal *See* Aprobarbital
- Oblivon *See* Methylpentynol
- 1-Octanol – **C** — Octyl alcohol
247, 611
- 2-Octanol – **C** — *n*-Octyl alcohol
247, 760, 1057
- Octyl alcohol *See* 1-Octanol
- n*-Octyl alcohol *See* 2-Octanol
- Olive oil – **C**
417, 847, 917
- Omeril *See* Mebhydrolin
- Opiates – **N**
460, 500, 520, 916, 985, 1239
- Opipramol – **P** — Insidon
377, 1196, 1343, 1498
- Opium – **C**
63, 439, 451, 636, 764, 785, 849, 850
- Optalidon – **N**
553
- Ortedrine *See* Amphetamine
- Ouabain – **C**
1421, 1444
- Oxalic acid – **C**
769
- Oxaloacetic acid – **N**
769
- Oxazepam – **R**
491
- Oxedrine tartrate – **C** — Sympathol
1413
- Oxytetracycline – **R** — Terramycin
366
- Oxytocin – **R** — Pitocin
276, 429, 430, 1459, 1460
- Pactal *See* Pecazine
- Pagitane hydrochloride *See* Cycrimine hydrochloride
- Palliopen – **N**
1066
- Palmitic acid – **C**
181
- Panax ginseng *See* Ginseng
- Pantocaine *See* Tetracaine
- Papaverine – **C**
322
- Paraffin oil *See* Petrolatum liquid
- Paraldehyde – **C**
9, 123, 175, 204, 301, 347, 351, 595, 665, 688, 693, 717, 718, 864, 994, 1075, 1289, 1311, 1442, 1483
- Paranitrochlorbenzol – **N**
254, 1063
- Para-sanol tablets – **N**
(*N*-diphenyl-methyl-atropine bromide + aluminium glycinate + meprobamate)
1498
- Parathion – **C**
23, 24, 1452
- Paredrine *See* Hydroxyamphetamine
- Pargyline – **R**
1100
- Passiflora – **C**
764
- Pecazine – **P** — Mepazine,
N-Methylpiperidyl-3-methylphenothiazine,
Pactal, Pekasin
188, 371, 373, 374, 396, 506, 714, 1067, 1453, 1481, 1498
- Pekasin *See* Pecazine
- Pemoline – **R** —
5-Phenyl-2-amino-4-oxo-oxazolidin, Tradone
569, 1401
- Penicillin – **C**
30, 65, 366, 690, 1365
- Pentamethazol *See* Pentetrazole
- Pentetrazole – **R** — Cardiazole, Corazol,
Metrazole, Pentamethazol, Pentylenetetrazol
15, 76, 159, 504, 559, 564, 574, 582, 674, 722, 730, 791, 839, 893, 950, 957, 983, 996, 1023, 1058, 1094, 1148, 1182, 1204, 1357, 1413, 1456, 1457
- Pentobarbital – **R** — Nembutal, Pentobarbital sodium, Pentobarbitone, Sodium pentobarbital
14, 15, 48, 155, 211, 261, 264, 304, 316, 337, 339, 340, 343, 360, 411, 426, 438, 443, 505, 519, 522, 534, 538, 561, 564, 591, 597, 600, 617, 633, 645, 646, 661, 662, 688, 702, 715, 756, 759, 792, 823, 825, 826, 827, 894, 969, 981, 1034, 1036, 1044, 1087, 1090, 1103, 1154, 1163, 1166, 1167, 1211, 1215, 1227, 1231, 1253, 1254, 1255, 1274, 1280, 1281, 1301, 1363, 1374, 1375, 1376, 1444, 1446, 1448, 1462, 1463, 1469, 1472, 1498
- Pentobarbital sodium *See* Pentobarbital
- Pentobarbitone *See* Pentobarbital
- Pentolinium tartrate – **C**
1447

- Pentothal *See* Thiopental sodium
- Pentothal sodium *See* Thiopental sodium
- Pentylene-tetrazol *See* Pentetrazole
- Pentymal *See* Amobarbital
- Perazine – **P** —
10-[3-(4-Methyl-1-piperazinyl)-
propyl]-phenothiazine, Taxilan
989, 990, 1498
- Percaïne *See* Cinchocaine chloride
- Perchlorethylene *See* Tetrachloroethylene
- Permitil *See* Fluphenazine
- Pernocton *See* Butallylonal
- Perphenazine – **P**
245, 517, 708
- Persantin *See* Dipyridamole
- Pertofran *See* Desipramine
- Pervitin *See* Methamphetamine
- Pethidine – **P** — Demerol, Dolantin, Mephedine
165, 631, 688, 858, 926, 1459, 1460, 1498
- Petrolatum liquid – **C** — Paraffin oil
78, 847
- Phanodorm *See* Cyclobarbital
- Phasein forte – **N**
(reserpine +
 β -bi-methylaminethyl-2-methylbenzhydriether
+ hydrochloride)
188, 396
- Phenacetin – **R** — Acetophenetidine
540, 664, 1089, 1203, 1397, 1398, 1408, 1498
- Phenactyl *See* Chlorpromazine
- Phenaglycodol – **R**
421, 423, 426, 495, 496, 600, 715, 804, 1034,
1498
- Phenamine
(transliteration)
See Amphetamine
- 1,10-Phenathroline – **C**
695
- Phenazone – **R** — Antipyrine
540, 776, 1117, 1210, 1211, 1214, 1215, 1498
- Phenbutamide – **R**
1156, 1157, 1158
- Phenbutazone *See* Phenylbutazone
- Phenelzine – **P** — Phenelzine sulfate
814, 882, 1396
- Phenelzine sulfate *See* Phenelzine
- Phenemal *See* Phenobarbital
- Phenergan *See* Promethazine
- Phenethyl alcohol – **C**
1422
- Phenformin – **P**
795, 796
- Pheniprazine – **P** — JB-516,
Phenylisopropylhydrazine
1190, 1273
- Phenmetrazine – **P** — Dexphenmetrazine,
Preludin
1296, 1405
- Phenobarbital – **R** — Gardenal, Luminal,
Phenemal, Phenobarbitone, Sodium
phenobarbital
23, 24, 25, 80, 92, 185, 187, 217, 219, 224,
225, 227, 261, 264, 336, 338, 344, 362, 406,
407, 451, 452, 453, 467, 468, 476, 491, 526,
531, 570, 581, 632, 633, 641, 646, 677, 684,
685, 686, 692, 706, 707, 757, 759, 783, 784,
785, 787, 818, 825, 826, 827, 835, 855, 861,
876, 882, 906, 923, 926, 983, 989, 1028, 1044,
1046, 1047, 1052, 1068, 1073, 1075, 1076,
1101, 1102, 1108, 1110, 1117, 1130, 1218,
1230, 1258, 1274, 1349, 1358, 1362, 1370,
1386, 1397, 1398, 1441, 1448, 1453, 1463,
1498
- Phenobarbitone *See* Phenobarbital
- Phenoglycodole – **N**
492, 493
- Phenol – **C** — Carboic acid
6, 354, 442, 462, 546, 670, 696, 737, 1060,
1061, 1072, 1135, 1415
- Phenothiazine – **R**
127, 133, 249, 429, 430, 432, 553, 718, 773,
1230
- Phenothiazine derivatives – **C**
188, 839, 1408
- Phenoxybenzamine – **R**
644, 648, 1267
- Phenoxymethylpenicillin – **R**
64
- Phenprocoumon – **R** — Marcoumar
1066, 1432
- Phentermine – **R** — Mirapront
1405
- 5-Phenyl-2-amino-4-oxo-oxazolidin *See* Pemoline
- 1-Phenyl-2-aminopropane *See* Amphetamine
- Phenylbutazoline – **N**
762
- Phenylbutazone – **R** — Butazolidin, *d*-Butazolidin,
Butazolidine, Phenbutazone
552, 553, 785, 1001, 1110, 1279, 1397, 1398,
1435, 1498
- o*-Phenylenediamine – **C**
737
- p*-Phenylenediamine – **C**
737
- Phenylethyl barbiturates – **N**
1026

Promethazine

- Phenylethylbarbituric acid – **N**
1028, 1066
- Phenylethylbiguanide – **N**
1189
- Phenylhydroxylamine – **C**
623
- Phenylisopropylhydrazine *See* Pheniprazine
- Phenylnorcamphan – **N**
969
- 1-Phenyl-2-pyrrolidylpentane *See* Prolintane
- Phenyltoloxamine – **R**
320
- Phenytol – **R** — Dilantin,
5,5-Diphenylhydantoin, Zentropil
630, 655, 658, 664, 957
- Phlorhizin *See* Phlorizin
- Phloridzin *See* Phlorizin
- Phlorizin – **C** — Phlorhizin, Phloridzin
241, 257, 626, 769
- Pholedrine sulfate – **R** — Veritol
538
- Phosphorus – **C**
242, 243, 354, 1011
- Physostigmine – **C** — Eserine
102, 194, 816, 818, 1419
- Picrotoxin – **C**
294, 539, 595, 730, 839, 845, 1090, 1448, 1456
- Pilocarpine – **C**
664, 816, 968, 1077, 1451
- Pipradol – **R**
664
- Pitocin *See* Oxytocin
- Pituitrin – **C**
143, 216
- Placebo – **N**
130, 320, 425, 428, 432, 441, 449, 494, 585,
587, 612, 641, 650, 676, 682, 684, 694, 754,
772, 873, 876, 878, 880, 881, 886, 927, 931,
945, 946, 1337, 1427, 1490, 1499
- Polamidone *See* Methadone
- Polyethoxydodecan *See* Thesit
- Polysan *See* Magnesium hydroxide
- Polysorbate 80 *See* Sorbimacrogol
- Porphyrin – **N**
1498
- Potassium antimonyl tartrate – **C**
71
- Potassium bromide – **C**
204
- Potassium chloride – **C**
122, 477, 648, 1170, 1325
- Potassium chromate – **C**
78
- Potassium cyanide – **C**
118, 399, 723, 769, 1256, 1257
- Potassium hyposulfite *See* Potassium thiosulfate
- Potassium iodide – **C**
895, 1171, 1484, 1485
- Potassium ions – **N**
1393, 1451
- Potassium metabisulfite – **C** — Potassium
pyrosulfite
1302
- Potassium pyrosulfite *See* Potassium metabisulfite
- Potassium rhodanate *See* Potassium thiocyanate
- Potassium thiocyanate – **C** — Potassium
rhodanate
247, 760, 1498
- Potassium thiosulfate – **C** — Potassium
hyposulfite
399
- Povidone – **P** — Subtosan
858
- Prednisolone – **R**
701
- Preludin *See* Phenmetrazine
- Presinol *See* Methyldopa
- Prethcamide – **C**
1498
- Prilocaine – **P** — L-67
1479
- Primidone – **P** — Mylepsin
630
- Proadifen – **P** — SK&F-525-A
677, 788, 792, 1131, 1386, 1486
- Probanthine *See* Propantheline bromide
- Procaine – **P** — Novocaine
221, 222, 302, 930, 1071, 1086, 1351, 1479
- Procaine hydrochloride – **C**
62, 502
- Procalmadiol *See* Meprobamate
- Prochlorpemazine *See* Prochlorperazine
- Prochlorperazine – **R** — Prochlorpemazine
582, 757
- Procyclidine – **R** — Kemadrin
773
- Prolintane – **P** — 1-Phenyl-2-pyrrolidylpentane
969
- Promazine – **P** — Veropen
127, 188, 357, 506, 547, 569, 582, 678, 1350,
1369, 1496, 1498
- Promethazine – **R** — Fargan, Phenergan
87, 338, 339, 340, 357, 495, 496, 621, 631,
678, 691, 797, 825, 826, 827, 858, 1154, 1169,
1259, 1486, 1492, 1498

- Promill-Ex – N
(yeast + fatty acids + caffeine extract)
775, 828, 865, 1458, 1481, 1490
- Propallylonal – P — Noctal
456, 1448
- Propanediol derivatives – N
1408
- Propanidid – P
329, 1216, 1217, 1492
- n*-Propanol *See* 1-Propanol
- 1-Propanol – C — *n*-Propanol, *n*-Propyl alcohol
138, 936, 937, 1054, 1175, 1433
- 2-Propanol – C — *i*-Propyl alcohol
5, 98, 138
- Propantheline bromide – R — Probanthine
1206
- d*-Propoxyphene *See* Dextropropoxyphene
- Propranolol – P
1267
- i*-Propyl alcohol *See* 2-Propanol
- n*-Propyl alcohol *See* 1-Propanol
- Propylene glycol – P
211, 776
- Propylthiouracil – R
580
- Prostigmine *See* Neostigmine
- Proteins – N
1260
- Prothipendyl – R — Dominal forte
188, 569, 761, 762, 1498
- Pseudotropine benzoate – C — Tropacocaine
101, 1010
- Psilocybine – R
762
- Psychotropics – N
694, 880, 884, 1404
- Psyquil *See* Triflupromazine
- Puromycin – P
34
- Pyramidon *See* Aminophenazone
- Pyrazinamide – C
530
- Pyrazine – C
693
- Pyrazole – C
151, 281, 497, 768, 776, 777, 788, 789, 921,
1168, 1178, 1344, 1436, 1437
- Pyrazole derivatives – N
776, 1408, 1498
- Pyrazolone – N
1498
- Pyrazolone derivatives – N
1118, 1402
- Pyridine – C
67
- Pyridine derivatives – N
1198
- Pyridine-2-aldoxime-dodecyl – N
1417, 1419
- Pyridoxine – P
775
- Pyridoxinium chloride – R — Vitamin B₆
375, 620, 1079, 1492
- Pyrilamine *See* Mepyramine
- Pyrocatechol – C
737
- Pyruvates – N
607, 618
- Pyruvic acid – C
769, 960
- Quadronal – N
(phenacetin + phenyldimethylpyrazolone)
1066
- Quinalbarbital *See* Secobarbital
- Quinhydrone – C
737
- Quinine – C
158, 567, 739, 849, 850, 995, 1022, 1134,
1389, 1453, 1481
- Quinine bisalicylosalicylate – C — Quinisal
456, 1448
- Quinisal *See* Quinine bisalicylosalicylate
- 8-Quinolinol – C — 8-Hydroxyquinoline
695
- Rastinon *See* Tolbutamide
- Raucombin D – N
(6-chlor-7-sulfamyl-3,4-dihydro-1,2,4-benzothia-
diazine-1,1-dioxide + rauwolfia alkaloids +
reserpine + hexamethonium bromide + rutin
+ potassium fluoride)
1066
- Reactivan *See* Fencamfamin
- Redoxon *See* Ascorbic acid
- Relaxa-tabs – N
1471
- Reserpine – R — Rivasin, Serpasil
82, 93, 105, 176, 177, 187, 188, 195, 248, 396,
421, 423, 426, 429, 430, 432, 516, 518, 597,
600, 648, 664, 676, 708, 715, 761, 762, 913,
947, 1089, 1109, 1169, 1230, 1301, 1321, 1349,
1369, 1473, 1498
- Resorcinol – C — Resorcin
362
- Resorcin *See* Resorcinol
- Resorcinol – C
737
- Restenil *See* Meprobamate

Sodium iodide

- Revonal *See* Methaqualone
 Rhodan combinations – N
 1081, 1498
 Rhodanate – N
 199
 Rimifon *See* Isoniazid
 Ritalin *See* Methylphenidate
 Rivasin *See* Reserpine
 Ro 5-4556 *See* 7-Chloro-2, 3-dihydro-1-methyl-5-phenyl-1H 1, 4-benzodiazepine hydrochloride
 Roter tablets – N
 (magnesium carbonate + bismuth subnitrate + sodium bicarbonate)
 1498
 Salidyston – N
 (2-benzyl-4,5-imidazoline salicylic acid + diethylaminoethyl-benzilate + phenylethylbarbituric acid)
 396
 Saline – N
 141, 187, 317, 435, 582, 644, 676, 905, 973, 974, 981, 1036, 1194, 1349, 1365, 1386, 1419, 1420, 1442, 1462, 1486, 1487
 Salyrgan *See* Mersalyl
 Saridon – N
 362, 552, 553, 584, 784, 1008, 1203, 1498
 SB-1 *See* 1-(*m*-Aminobenzenesulfonyl)-3-butylurea
 Scophedal – N
 (scopolamine hydrobromide + oxycodone + racephedrine)
 1029, 1071, 1076
 Scopolamine – C
 356, 620, 907, 908, 909, 910, 958, 959, 1071, 1154, 1276, 1459, 1460
 Secbutabarbital – R — Butabarbital
 326, 445, 460, 591, 1026, 1028, 1291
 Secobarbital – R — Immenoctal, Quinalbarbital, Secobarbital sodium, Secobarbitone, Seconal
 77, 92, 155, 398, 443, 526, 534, 759, 804, 862, 863, 999, 1073, 1090, 1440, 1448, 1459, 1469
 Secobarbital sodium *See* Secobarbital
 Secobarbitone *See* Secobarbital
 Seconal *See* Secobarbital
 Sedamyl *See* Acecarbromal
 Seda-tabs – N
 1471
 Sedatives – N
 30, 349, 358, 359, 403, 416, 436, 500, 619, 628, 673, 680, 683, 765, 807, 869, 1025, 1029, 1100, 1111, 1162, 1164, 1288, 1297, 1400, 1401, 1402, 1403, 1404, 1406, 1408, 1429, 1498
 Sedormid *See* Apronal
 Seduan – N
 285
 Serotonin – C — 5-Hydroxytryptamine
 93, 1149, 1332
 Serpasil *See* Reserpine
 Sidol – N
 1071
 SK&F-525-A *See* Proadifen
 Snake and other venoms – C
 84, 260, 271, 334, 439, 458, 484, 508, 589, 590, 727, 793, 856, 895, 917, 949, 951, 993, 1013, 1030, 1063, 1347, 1373, 1485
 Sodium acetate – C
 816, 1325, 1359
 Sodium acetylsalicylate – N
 772
 Sodium amytal *See* Amobarbital
 Sodium arsenite – C
 607, 716
 Sodium azide – C
 788
 Sodium barbital *See* Barbital
 Sodium benzoate – C
 36, 620, 1234
 Sodium bicarbonate – C
 163, 251, 257, 379, 1015, 1123, 1142, 1145, 1242, 1365, 1492
 Sodium bromide – C
 540, 1345
 Sodium chlorate – C
 310
 Sodium chloride – C
 353, 477, 691, 718, 877, 1287, 1366
 Sodium citrate – C
 816
 Sodium cyanide – R
 788, 1057, 1131
 Sodium diethyldithiocarbamate – C
 16, 720
 Sodium dihydrocholine – N
 1224
 Sodium ethyl phosphate – N
 816
 Sodium fluoride – C
 1287
 Sodium formate – C
 90
 Sodium hexobarbital – N
 1446
 Sodium hydroxide – C — Caustic soda
 735, 739
 Sodium iodide – C — Iodine natrium
 1240

- Sodium iodoacetate – **N**
243
- Sodium levothyroxine – **R** — Thyroxine
216, 457, 557, 674, 746, 1100, 1453
- Sodium malate – **C**
816
- Sodium maleate – **N**
819
- Sodium nicotinate – **C**
1358
- Sodium nitrite – **C**
265, 623
- Sodium oxalate – **C**
1287
- Sodium pentobarbital *See* Pentobarbital
- Sodium phenobarbital *See* Phenobarbital
- Sodium phenyl ethylbarbiturate – **N**
722
- Sodium phenylbutazone – **N**
664
- Sodium phytate – **P**
1287
- Sodium pyruvate – **N**
816, 1271, 1325
- Sodium rhodanide *See* Sodium thiocyanate
- Sodium salicylate – **C**
36
- Sodium succinate – **C**
16
- Sodium sulfate – **C**
95
- Sodium thiocyanate – **C** — Sodium rhodanide
784, 785, 1498
- Sodium veronal *See* Barbital
- Somnifene *See* Diethylamine barbiturates
- Somnin – **N**
(barbituric acid +)
784, 785, 1052, 1453
- Soneryl *See* Butethal
- Soprontin *See* Acepromazine maleate
- Sorbimacrogol – **R** — Polysorbate 80
779
- Sorbit *See* Sorbitol
- Sorbitol – **C** — Sorbit
1383
- Soventol *See* Bampine
- Spasmo-cibalgin – **N**
(dimethylaminophenazone +
dimethylaminophenazone-diallylbarbituric acid
+ chlorhydrate of
hexahydro-diphenyl-acetyldiethylaminoethanol
ester)
1498
- Spasmolytics – **N**
1498
- Sterisocaine – **N**
1066
- Stimulants – **N** — Weckamine
30, 53, 54, 167, 271, 349, 431, 436, 460, 520,
680, 807, 928, 1034, 1070, 1078, 1283, 1298,
1301, 1400, 1404, 1429
- Stop – **N**
(coffee extract + dextrose + ammonium
carbonate + vitamin B₁ + sodium carbonate
+ chlorophyllin + magnesium azulen)
374, 734, 775, 1040, 1481
- Streptomycin – **R**
366, 530, 693
- Strophanthin – **C**
1480
- Strychnine – **C**
31, 73, 74, 173, 274, 323, 325, 353, 435, 458,
461, 489, 490, 540, 548, 549, 560, 577, 613,
615, 616, 627, 674, 730, 791, 808, 849, 853,
859, 860, 918, 922, 983, 1019, 1045, 1063,
1134, 1135, 1172, 1287, 1294, 1331, 1373,
1484
- Subtosan *See* Povidone
- Succinate – **N**
608, 1325
- Succinic acid – **C**
236, 769
- Succinylcholine iodide – **C** — Suxamethonium
iodide
1419
- Sufrogel – **N**
1391
- Sugars – **N**
966, 1041, 1073, 1074
- Sulfadiazine – **R**
962
- Sulfaguanidine – **R**
826, 827
- Sulfamethoxydin *See* Sulfametin
- Sulfametin – **P** — Sulfamethoxydin
654
- Sulfanilamide – **R**
635, 820, 1157, 1211, 1212, 1213, 1229, 1287
- Sulfonamide – **C**
29, 133, 784, 785, 1498
- Sulfonylurea preparations – **N**
42, 332, 349
- Suprarenin *See* Adrenaline
- Suprifen *See* *p*-Hydroxyephedrine
- Suxamethonium iodide *See* Succinylcholine iodide
- Sympathol *See* Oxedrine tartrate

Tranlycypromine

- Tacitin – **N**
640
- Tannic acid *See* Tannin
- Tannin – **C** — Tannic acid
853, 954, 1243
- Targesin – **N**
(colloidal complex diacetylaminosilveralbumin combination)
1498
- Taxilan *See* Perazine
- Tegretol *See* Carbamazepine
- Terramycin *See* Oxytetracycline
- Tertiary amines – **N**
54
- Tetracaine – **R** — Pantocaine
101, 1479
- Tetracemin *See* Edetic acid
- Tetrachlorates – **N**
1293
- Tetrachloroethylene – **C** — Perchlorethylene
272
- Tetracycline – **R**
64
- Tetraethylammonium bromide *See*
Tetrylammonium bromide
- Tetraethyllead – **C**
124, 1080, 1414
- Tetraethylthiuram disulfide *See* Disulfiram
- Tetrahydrocannabinol – **C**
837
- Tetramethylthiuram disulfide *See* Thiram
- Tetramethylthiuram monosulfide – **N**
16, 60, 544, 695
- Tetrapon – **P**
37
- Tetrylammonium bromide – **R** —
Tetraethylammonium bromide
664
- Thalidomide – **P**
1290
- THAM buffer *See* Trometamol
- Theophylline – **C**
1055
- Thesit – **C** — Polyethoxydodecan
1479
- Thialbarbital – **R** — Kemithal
1188, 1448
- Thiamine – **P**
816
- Thiamine chloride – **N**
876
- Thiamine tetrahydrofurfuryl disulfide – **C**
129
- Thiobarbiturates – **N**
929
- Thiobutabarbital – **N**
328, 445, 521, 1291
- Thiocarbanilide – **C**
16
- Thiocyanates – **N**
121, 683, 1498
- Thiopental – **R**
261, 264, 328, 356, 521, 766, 785, 1087, 1188,
1215, 1216, 1217, 1270, 1277, 1448
- Thiopental sodium – **R** — Pentothal, Pentothal
sodium, Thiopentone, Trapanal
112, 342, 464, 527, 693, 858, 877, 994, 1090,
1092, 1154, 1231, 1274, 1443, 1448, 1463
- Thiopentone *See* Thiopental sodium
- Thioridazine – **R**
757, 762, 882, 883, 887, 888
- Thiram – **P** — Tetramethylthiuram disulfide
16, 60, 544, 555, 689, 695, 720, 1238, 1299,
1385, 1498
- Thomapyrine *See* APC
- Thorazine *See* Chlorpromazine
- Thymeretics – **N**
1229, 1498
- Thymoleptics – **N**
1229, 1498
- Thymyl isoamylcarbamate – **C** — Egressin
753, 866, 1498
- Thyroxine *See* Sodium levothyroxine
- Tobacco *See* Nicotine
- Tofranil *See* Imipramine
- Tolboxane-R-Clarmil
248
- Tolbutamide – **R** — D-860, Rastinon
17, 42, 61, 105, 198, 200, 201, 208, 231, 286,
643, 655, 656, 657, 658, 666, 749, 824, 962,
963, 1064, 1100, 1157, 1158, 1230, 1364, 1498
- Tolserol *See* Mephenesin
- Toluene – **C**
1307
- Tolycaine – **P** — Baycain
1479
- Tradone *See* Pemoline
- Trancylpromine – **R** — Tranlycypromine
33, 268, 432, 1099
- Tranquilizers – **N**
30, 53, 68, 167, 214, 239, 246, 330, 347, 351,
416, 432, 460, 520, 619, 628, 680, 683, 765,
869, 887, 985, 987, 1070, 1100, 1161, 1219,
1229, 1261, 1262, 1298, 1400, 1406, 1429,
1441, 1498
- Tranlycypromine *See* Trancylpromine

Trapanal

- Trapanal *See* Thiopental sodium
 Trepidone *See* Mephenoxalone
 Tribromethanol *See* Bromethol
 1,1,1-Trichloroethane – **C**
 272
 Trichloroethanol – **C**
 475, 1033
 Trichloroethylene – **R**
 23, 24, 106, 107, 113, 114, 272, 738, 758, 799,
 1193
 Triethyl orthoformate – **C** — Ethyl formate
 936, 937, 1175
 Trifluoperazine – **P** — Jatroneural, Trifluperazine
 882, 887, 1024, 1498
 Trifluperazine *See* Trifluoperazine
 Triflupromazine – **P** — Psyquil
 1203, 1498
 3,5,3'-Triiodothyronine *See* Liothyronine
 Trimethadione – **R**
 957
 Trimetozine – **P** — Trioxazine
 592
 Trimipramine – **P**
 878, 882, 887
 Trinitrin *See* Glyceryl trinitrate
 Trinitrotoluene – **C**
 1063, 1340, 1341
 Trioxazine *See* Trimetozine
 Tripelennamine *See* Tripelennamine hydrochloride
 Tripelennamine hydrochloride – **R** —
 Tripelennamine
 87, 495, 496, 604
 Triton *See* Tyloxapol
 Trometamol – **R** — THAM buffer
 1123
 Tropacocaine *See* Pseudotropine benzoate
 Truxal *See* Chlorprothixene
 Trypan blue – **C**
 721, 820
 Tryptizol *See* Amitriptyline
 Tuberculostatics – **N**
 1203, 1498
 Tubocurarine chloride – **R** — Curare
 1154, 1170, 1419
 Tuinal – **N**
 (amobarbital + secobarbital)
 155, 196, 1498
 Tutocaine *See* Butamin
 Tyloxapol – **R** — Triton
 1419
 Tyramine – **C**
 1149, 1329
 Uranium nitrate – **C**
 242, 243
 Urethane – **R** — Ethyl urethane
 216, 300, 524, 543, 825, 1087, 1092, 1431
 Valepotriates – **N**
 364, 365
 Valerian – **C**
 764
 Valium *See* Diazepam
 Valmane – **N**
 365
 Valtratum – **N**
 364
 Vasopressin – **P**
 160, 276
 Veramon *See* Barbipyrine
 Veritol *See* Pholedrine sulfate
 Verla-3-dragées – **N**
 1005, 1006
 Veronal *See* Barbitol
 Veronal sodium *See* Barbitol
 Verophen *See* Promazine
 Vesparax – **N**
 (atarax + barbituric acid derivatives)
 285, 1066
 Vinegar – **N**
 1348
 Vinylbital – **R**
 761, 762
 Viomycin – **P**
 530
 Vitamin A – **P**
 1469
 Vitamin B complex – **N**
 688, 760, 966
 Vitamin B₆ *See* Pyridoxinium chloride
 Vitamin B₁₂ *See* Cyanocobalamin
 Vitamin C *See* Ascorbic acid
 Vitamins – **N**
 30, 185, 582, 723, 886, 933, 1041, 1079, 1297,
 1469
 Warfarin – **C**
 61, 655, 656, 658
 Weckamine *See* Stimulants
 Wine – **N**
 3, 44, 88, 104, 115, 117, 141, 186, 199, 212,
 240, 245, 260, 306, 336, 367, 371, 456, 461,
 483, 510, 533, 541, 594, 606, 681, 687, 694,
 697, 698, 699, 700, 739, 745, 778, 798, 847,
 911, 919, 946, 978, 1004, 1012, 1040, 1066,
 1192, 1198, 1200, 1203, 1234, 1235, 1371,
 1388, 1389, 1401, 1427, 1434, 1448, 1453,
 1461, 1481
 Xanthine – **C**
 1272

Xanthoxylum – C

153

Xylose – C

1260

Zentranol – N

(diphenylhydantoin + luminal)

1230

Zentropil *See* Phenytoin

Zinc diethyldithiocarbamate – N

16

Zoxazolamine – R

561

Zyanamid *See* Cyanamides

PLEASE DO NOT REMOVE
CARDS OR SLIPS FROM THIS POCKET

UNIVERSITY OF TORONTO LIBRARY

Z Polacsek, Eric Paul
7721 Interaction of alcohol
P58 and other drugs
1972

BioMed

